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THE PATHOLOGICAL EFFECTS OF RADIANT ENERGY ON THE EYE

AN EXPERIMENTAL INVESTIGATION

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WITH A SYSTEMATIC REVIEW OF THE LITERATURE

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FROM THE PATHOLOGICAL LABORATORY OF THE MASSACHUSETTS CHARITABLE EYE AND EAR INFIRMARY.

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THE fundamental purpose of this investigation has been to discover what if any pathological effects can be produced upon the structure of the eye by exposure to artificial or natural sources of light. That such action may occur under sufficiently powerful exposure to radiant energy is certain, but the essential fact is the discovery of the quantitative relations between the amount of incident energy and the effects. These relations have generally been left quite out of the reckoning in discussing the subject, with the result of leading to vague and often quite unwarranted conclusions as irrelevant as if one should condemn steam heating as dangerous because one can burn his finger upon a radiator.

The quantitative phase of the matter is, from a practical standpoint, all-important since on it depends the actual effects to be expected from the exposure of the eye to powerful natural or artificial sources of light. Although the literature of the subject is very extensive, the appended bibliography covering over 450 titles, practically none of the work done has been quantitative in the sense of connecting the amount and kind of the energy received with the effects produced, and hence, despite the work of many careful investigators, the results have been singularly discordant and inconclusive, so that a coördination of the facts from the standpoint of energy has seemed imperative. One of us [19] has investigated recently the energy relations of the radiation from various sources of light both natural and artificial, and the intent of the present investigation has been to determine by actual experiment on the eye the quantitative and qualitative effects of radiant energy on the conjunctiva, cornea, iris, lens, and retina.

It has long been known that excessive radiation of one kind or another produces pathological changes in the eye, of many kinds and greatly varying degrees of intensity. So far as natural light is concerned the well known effects of powerful solar radiation in producing snow blindness and allied troubles have been long familiar as also have been the severe scotomata due to direct observation of the sun, familiar in the literature under the general name of eclipse blindness. With the introduction of the electric arc, mild cases of ocular trouble due to over exposure to the arc began to attract attention, at first nearly a half a century ago, and the subject has occupied an increasing space in the literature ever since. More recently attention has been particularly drawn to the ultra violet radiation as productive of these pathological conditions, and most of the investigations bearing on the general subject have been directed toward the study of the specific

action of the ultra violet. It, therefore, becomes of fundamental importance to examine the effects of radiant energy with special reference to their relation to the wave length of the radiation.

Nature and Distribution of Radiant Energy.

All radiant energy is at present believed to consist of transverse vibrations in the hypothetical ether, all propagated at the same rate and differing only in amplitude and wave length, hence in frequency, which is the reciprocal of wave length. The uniform propagation rate in vacuo is very nearly 300,000 km. per second and the wave lengths so far as ordinarily dealt with range from about .01 to about .0002 mm. For ordinary purposes no attention need be paid to the extremely long wave lengths ranging to .1 mm., to the extremely short ones between .0001 and .0002 mm., or to the enormously shorter one still of the order of magnitude of .0000001 mm. such as the X-rays are believed to be. For the very long waves are not present in material amount in the radiation from ordinary sources. The very short ones are absorbed by a few cm. or dm. of air, and the X-rays are practically only produced in apparatus intended for that purpose. The spectra given by all ordinary sources range between the more modest limits just given. In the earlier literature this spectral range used to be divided into heat rays, light rays, and actinic rays, a distinction wholly artificial since the three effects implied are far from being sharply defined. More generally the whole range is divided into the infra red portion, not ordinarily visible and extending from the longest waves to those of about 760 $\mu\mu$, the visible spectrum, extending from about 760 $\mu\mu$ to about 395 $\mu\mu$, and the ultra violet portion reaching from 395 $\mu\mu$ to the neighborhood of 200 $\mu\mu$. This distinction is not rigorous or with sharp limits. While artificial distinctions have led to many misunderstandings, all radiation of whatever wave length is convertible into heat when absorbed by material bodies and may produce chemical changes as well. As a matter of fact these latter show a general tendency to increase with the frequency of the oscillations, so that chemical changes are rare in the infra red and increasingly frequent as one approaches the extreme ultra violet. It is this tendency that is shown in the pathological changes which may be caused in living cells by the incidence of radiation.

The rationale of the chemical effect of radiation seems to be that while all radiation transfers energy to the molecules which absorb it

and produce heat, certain particular wave frequencies fall into step, as it were, with the oscillation periods which depend on the molecular structure, and so break up the molecules when the energy absorbed is sufficient. The particular kind of radiation which produces this direct action depends on the character of the molecules. Thus, for instance, the green modification of silver bromide is readily broken up by radiation of wave length as great as 1 μ, while it requires radiation of double this frequency to affect ordinary silver bromide, and the molecules of living protoplasm begin to break up only when the wave length is down to about 300 $\mu\mu$ as we shall show. But most chemical compounds are unaffected by any practical amount of radiation which may fall upon them except as they may be heated to the point of decomposition. Any effect which is due to radiation is in the last analysis dependent on the absorption of that radiation, in that there is involved a transfer of energy to the molecules or their parts in order that they may be heated or shaken apart. Every substance absorbs radiant energy in greater or less degree, and the amount of absorption bears a definite relation to the thickness of the body as well as to the particular wave length of the incident energy. Certain substances, like fluorite and to a less degree quartz, let pass with very little obstruction radiation from far into the infra red to wave length 200 $\mu\mu$. Water absorbs the longer wave lengths of the infra red up to about 1.2 μ powerfully, and transmits nearly everything else up to the extreme ultra-violet, while pure air, generally speaking extremely transparent, produces some small but sharp absorption in the visible spectrum and completely wipes out the extreme ultra violet. But whatever the wave length, the law connecting the absorption of energy with thickness of the medium is extremely definite. If a layer of unit thickness transmits a certain fraction T then any other thickness x will transmit a fraction T^x of the incident energy. Thus, if a substance transmits badly, leading to a low value of T, very little energy gets through the outermost layers, while if it be fairly transparent a considerable amount of energy penetrates deeply. For example, a certain Jena glass transmits violet light through 1 mm. of thickness with only a small fractional per cent of loss. It transmits the same light through a cm. of thickness with the loss of only 2%, while near the extreme ultra violet of the solar spectrum it still transmits a little over 90% for a mm. of thickness, but barely 38% through a cm. Where, therefore, radiant energy falls on a solid upon which through absorption it produces powerful chemical action, the immediate effect will be almost wholly superficial, and only by prolonged and intense

radiation can enough energy be communicated into interior layers to affect them in a similar manner. To put the thing in another way, it is only relatively inactive rays that through consequent lack of absorption penetrate a medium easily. With sufficient incident intensity, however, enough energy may penetrate the outer layers to produce definite action within them.

In ordinary transparent media the loss of energy by real absorption in the substance is less than the loss at the surfaces by reflection. This loss depends on the refractive index of the medium with respect to the entering or emerging ray, and for nearly normal incidence the coefficient of reflection, that is the proportion of the ray transmitted through a single reflecting surface is $K = \frac{(n-1)^2}{(n+1)}$. This coefficient is the same for each successive surface of transition, so that for m surfaces the coefficient of transmission are $K_m = K^m$. Thus, if a glass plate has an index of refraction n the light transmitted is K^2

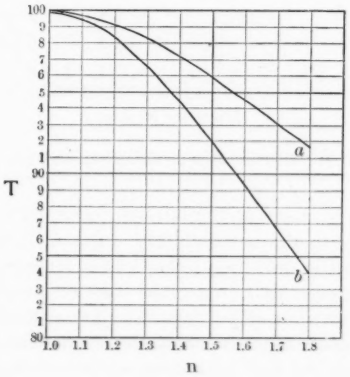


FIGURE 1. Transmission of glass surfaces.

for the two surfaces. In case of an optical system having several lenses, the reflection losses may be severe, particularly if some of the glasses are of high index. Figure 1 shows in curve a the transmission of a single surface for various indices of refraction, and in curve b transmission of a double surface like that presented by a transparent

plate. The reflection is a function as well of the angle of incidence, but for the ordinary angles up to 30 degrees or so the variation is negligible. At large angles of incidence such as would be presented, for instance, by the marginal rays of a beam incident upon the cornea the loss by reflection may be considerably more than doubled so as to materially reduce the amount of energy absorbed.

In any case the surface density of the energy received by the cornea

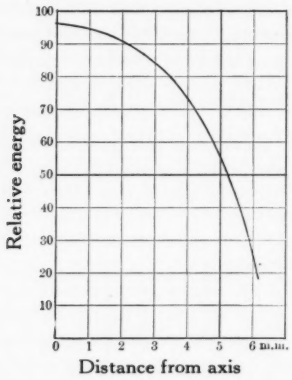


FIGURE 2. Distribution of radiant energy on cornea.

under such circumstances is diminished, following Lambert's law, in direct proportion to the cosine of the angle of incidence. The net result is that from parallel rays the cornea receives a much greater incidence of energy per unit area in the centre than toward the margin, which accounts for some of the results to be recorded later. Figure 2 shows for the average rabbit's cornea the approximate variation in the intensity of energy per unit area from centre to periphery.

PHOTOPHTHALMIA.

Inasmuch as most of the pathological changes in the eye observed, after exposure to light, either clinically or experimentally, have been ascribed to the action of the ultra violet part of the spectrum, it is with this that our work has chiefly been done, although we have also

examined the effect of the other radiations which are received from natural or artificial sources.

Our first aim was to ascertain what quantitative relations existed between the incidence of energy on the eye and the pathological effects which might follow. Especially we desired to ascertain whether these effects were proportional to the incident energy and hence to such primary lesions as might be produced by it, or serve to set in train pathological changes of an extent not proportionate to the primary inducing cause. To this end we first turned our attention to the so-called ophthalmia electrica or photophthalmia (Parsons), at once the earliest known and commonest of the superficial pathological effects of radiation. Probably first observed by Foucault and Despretz about sixty years ago, it received its first notice from the medical standpoint in a paper by Dr. Charcot.[66] His brief clinical observations are here reproduced in full as they are typical so far as the external effects go of a mild case of this particular affection. The luminous effects as described, are not characteristic and were no doubt purely psychical, and due perhaps to undue attention having been called to the sensations of light normally arising in the dark adapted eye. The fusion and vitrification of refractory substances produce far more intense effects of this kind than would have been noted in the other experiments cited by Dr. Charcot.

Erythema Produced by the Action of the Electric Light. By Dr. Charcot.— The fourteenth of February last two chemists were coöperating in making some experiments on the fusion and vitrification of certain substances by the action of the electric battery. They made use of a Bunsen battery of 120 elements. The experiments lasted about an hour and a half; but during this time the action of the battery was frequently interrupted and it was not working in all more than twenty minutes. At the distance of the experimenters from the arc, about fifty cm., they were not sensible of a rise in temperature. Nevertheless, that evening and during the whole night which they passed without sleep they found in their eyes a feeling of severe irritation and saw almost continually flashes and colored spots. The next day both had upon their faces erythema of a purplish color with a feeling of pain and tension. In the case of M. W., the right side of whose face alone was exposed to the luminous arc, the reddening covered that whole side from the roots of the hair to the chin, and the sparks were only seen as if before his right eye. In the case of M. M. who had held his head lower and whose face had been protected against the arc by his brow, the brow only was affected with erythema. Upon both the experimenters the appearance of the skin in the parts affected was exactly that of sunburn; a slight desquamation was established at the end of four days and lasted in all five or six days.

This effect of the electric light is very curious and in its pathology one may perhaps find the rationale of sunburn properly so-called. Everybody knows a high temperature is not a necessary condition for the production of this last affection, for there are some people who are attacked in the cool weather of the first days of spring, a fact analogous to those which we here report. Both concur in showing that in the radiation of the light it is not the calorific rays which attack the skin.

Must one then invoke the action of the luminous rays themselves? No, or at least the intensity of the light seems to play only a secondary rôle. Indeed in the experiments made by M. Foucault in coupling several Ruhmkorff coils to produce sparks of which the length increased with the number of bobbins and where he had been able by the means of a double action interrupter to double the number of these sparks without diminishing their energy, this observer was attacked by headache, very marked and persistent troubles of vision and erythema, although the light was not more intense than that of a star which one looks at without fatigue. M. Despretz has noted that light obtained with 100 Bunsen elements produces eyeache and that from 600 elements very rapidly produces erythema.

There remain the so-called chemical rays and it is this sort of rays which seems to be the principle essential agent of the accidents. To protect the eyes, it suffices as M. Foucault has several times noted, to let the electric light pass through a uranium glass screen which absorbs a large proportion of the chemical rays. Doubtless by protecting the face with this same uranium glass one would avoid also the production of erythema. The very rapid and energetic action of the electric light upon the skin and upon the retina one can understand the better since the chemical rays in it are as is well known relatively more abundant than in the solar light.

An ordinary clinical case of photophthalmia as observed after exposure to arc lights, short circuits, and the like, commonly takes the following course. After a period of latency, varying somewhat inversely with the severity of the exposure, but usually several hours, conjunctivitis sets in accompanied by erythema of the surrounding skin of the face and eyelid. There is the sensation of foreign body irritation, more or less photophobia, lacrimation, and the other ordinary symptoms of slight conjunctivitis. Occasionally there is some chemosis. The symptoms usually pass off in two or three days, and in severe cases there may be desquamation of the affected epidermis around the eye. In a very few instances the cornea has been slightly and temporarily affected. There is almost always immediately following the exposure, and quite unconnected with the photophthalmia proper, the ordinary results of a glare of light in the eyes, persistent after images, occasional scotomata, erythropsia and xanthopsia.

The diagnosis and prognosis we can hardly better state than in the words of Van Lint [381] in his report to the Belgian Ophthalmological Society.

"Diagnosis: The symptoms and evolution of the malady characterize very clearly accidents provoked by electric light. Nevertheless, the diagnosis is sometimes delicate. Certain people, especially employees working habitually by electric light, complain of ocular troubles which they assign to the influence of electricity. These troubles have for the most part no relation to the cause invoked. In the patient affected by conjunctivitis one generally finds a slight infective conjunctivitis, if by troubles of vision one finds asthenopia due either to a local cause, hypermetropia or astigmatism, or to a general cause anemia, fatigue or the like. One must consequently eliminate all these outside causes before concluding that the troubles are chargeable to the electric light."

"Prognosis: As one is able to see after a study of the symptoms the prognosis is always favorable. A duration of about five days seems to be necessary for the course of the malady. In case of nervous asthenopia the prognosis is equally favorable provided one protects the patient against the luminous sources. A case cited by Féré endured six weeks, but the patient was affected by nervous symptoms which had very remote relation with those provoked by electric light."

Our first series of experiments was concerned with the relation between cause and effect in photophthalmia of rabbits following exposure to a powerful source of ultra violet radiation. As the source of energy we employed a quartz mercury lamp operating on 220 volt circuit and normally taking 3.5 amperes with about 90 volts across the terminals of the tube. This was the same lamp of which the radiation has already been studied by one of us [19] and which furnishes by far the best source of energy for such experiments, inasmuch as its ultra violet radiation is powerful and the light after running twenty to thirty minutes to heat up is extraordinarily steady. It is also remarkably advantageous in the distribution of energy in its spectrum, since it gives off relatively little radiation of long wave length, the nearer infra red region being particularly weak, so that results obtained by it are not complicated, save in some experiments with bacteria, by any effects due purely to temperature. Although there is considerable heat loss in the lamp it is nearly all in the form of heat waves of very long wave length which are wholly cut off by a cell containing pure water, the infra red lines of the spectrum being very few. As respects the radiation from this lamp, therefore, it is practically all

in the visible and ultra violet portions of the spectrum, 35% being in the visible spectrum itself and 65% in the ultra violet between wave lengths 400 $\mu\mu$ and 200 $\mu\mu$. This 65% is equally divided between wave lengths 400 $\mu\mu$ to 300 $\mu\mu$ and 300 $\mu\mu$ to 200 $\mu\mu$, as one of us has already shown (loc. cit.). As the lamp was run the total radiation of energy having wave lengths less than 400 $\mu\mu$ at a distance of 50 cm. from the tube was to a very close approximation 11,000 ergs per second per square cm., of which 5,500 ergs per square cm. of energy were of wave length less than 300 $\mu\mu$. At distances other than the standard one here noted, the radiation follows the law of inverse squares with substantial precision. A small correction should theoretically be applied because the radiating body is approximately a cylinder instead of a point. But for all practical purposes this correction may be neglected, since for a radiative body of the dimensions used it amounts to less than one quarter of 1% at all distances greater than 50 cm., and does not exceed 2% even when the distance is reduced to 20 cm. Plate 5, Fig. 1, shows the actual spectrum of the quartz lamp taken with a rather wide slit and prolonged exposure with wave length scale annexed, and Figure 2 shows the stronger lines of the visible spectrum alone. The lines toward the right of A, which do not appear in B, are merely the ultra violet lines of the overlapping second order spectrum, the photograph having been taken with a concave grating of 1 meter focal length and 10,000 lines to the inch. Attention should be called to one interesting feature of this mercury arc spectrum. It will be seen that there is but a single ultra violet line between the strong double line at 313 $\mu\mu$ and the strong group at 365 $\mu\mu$ and this line is relatively weak. There is also a conspicuous gap between 313 and the next line at approximately 303 $\mu\mu$. These gaps in the spectrum are of some significance in interpreting bactericidal experiments in this region of the spectrum.

Determination of Liminal Exposure.

As a starting point in our experiments it was necessary to determine, using the standard source just described, how long exposure at some known distance was necessary in order to produce clearly marked symptoms of photophthalmia. Our experimentation throughout the work has been chiefly with rabbits, since these animals have been generally used by other experimenters and the characteristics of their eyes have therefore become fairly well known.

The method of experimenting was as follows: The animal was enclosed in a box without a cover through one end of which the head protruded, being held in place by a sliding end piece. The eye was held open by a Murdoch speculum made of proper size for the purpose, and was then exposed at a known distance from the quartz lamp working at standard intensity for a given period. A few hours later, usually the next day, after the course of the experiments was settled, careful examination was made of the external eye for any signs of effect from the radiation, the unexposed eye being used as a check. After a few preliminary trials we found that the occurrence of slight conjunctivitis was less readily determinable than the damage to the corneal epithelium showing in the reflection of light from the cornea by a slight irregular crackled appearance giving way after stronger exposures to faint stippling. A still greater severity of exposure produces faint haziness. We also tried staining with fluorescine as index of damage to the epithelium, but found in the first stages disturbance of the corneal light reflection a more reliable guide. This indicates a somewhat more severe exposure than produces the first trace of conjunctivitis, but its presence or absence is quite definite whereas the conjunctivitis may not be easy to determine if the rabbit's eyes are naturally somewhat reddened.

The minimum exposure at .5 meter required to produce the first signs of pathological change on the surface of the cornea was determined to be six minutes. The following experiments show the results leading to the establishment of this minimum, and it will be seen that the liminal period was sometimes about a minute shorter or longer, various animals differing somewhat in sensitiveness. It will be observed that to produce loss of corneal epithelium required an exposure about $2\frac{1}{2}$ times that necessary to produce slight photophthalmia.

Experiments.

Negative results. Exposures in minutes: 1; $2\frac{1}{2}$; 3; 3; 3; 5; 5; 5; $7\frac{1}{2}$.

Slight but definite conjunctivitis with impairment of corneal light reflection. Exposures in minutes: $7\frac{1}{2}$; 5; $7\frac{1}{2}$; $7\frac{1}{2}$; 6; 6; 6; $7\frac{1}{2}$; 6; 4 (albino).

Marked conjunctivitis with slight haze of cornea but without loss of corneal epithelium. Exposures in minutes: 10; 10.

Marked conjunctivitis with haze of cornea and loss of corneal epithelium. Exposure in minutes: 15.

Verification of Law of Inverse Squares.

The outcome of this series of experiments was that radiation from the mercury vapor lamp to the amount of 4×10^6 erg-seconds per square cm. is required to set up the first definite symptoms of photophthalmia. This assumes that the effect is proportional to time, in other words, that the pathological results are determined by the total amount of energy, and the next series of experiments was directed to the establishment of the truth or falsity of this assumption. For this purpose, having ascertained the liminal exposure for a single distance, .5 meter, exposures were made at various distances for times computed for equal total radiation, assuming the law of inverse squares to hold for the relative intensities. For example, at 1 meter the time required to produce the determining symptoms, assuming the law of inverse squares, should be four times that required for .5 meter, which was found to be closely the case. By repeated experiments at distances varying from about 20 cm. to 2.5 meters, the inverse square law was verified over a range of radiation intensities in the ultra violet varying from 72,000 ergs per square cm. per second down to 455 ergs per second per square cm., at a range in other words of 156 to 1. For any source yielding rays capable of producing pathological effects on the cornea therefore, the exposure time required to produce symptoms of photophthalmia is inversely proportional to the intensity of the radiation of such rays, and can be definitely determined when the intensity of the damaging radiation is known, subject to the condition that if the computed time reaches many hours it may be even further lengthened by the intervention of physiological repair. This conclusion is of fundamental importance, since it shows that the symptoms are due to or are proportional to, the direct and primary effect of the energy. Since, as we shall show later, the rays which are able to injure cells by chemical action are only those of wave lengths below 305 $\mu\mu$, these present experiments of ours show that the critical amount of such radiation required to set up well marked photophthalmia is approximately 2×10^6 erg-seconds per square cm. In other words only half of the total ultra violet already specified is effective in producing such symptoms.

A close general relation between the amount of incident energy and its effects on the cornea was beautifully shown by the results obtained after relatively severe exposures. In such cases after the symptoms had developed there was a distinct haziness confined chiefly to the

central portion of the cornea and rapidly shading off toward the periphery, where, as is shown in Figure 2, the energy received per unit area is greatly decreased. Thus the mere appearance of the affected area shows in a qualitative way proportionality between the exposure and the following lesion, a proportionality shown to be definite in the experiments we have described.

The Effects of Repeated Exposures to Abiotic Radiations.

A natural corollary of the proposition that the pathological effects of abiotic rays on the cornea are proportional to the energy is that at least for brief intermissions the effects of repeated short exposures are equivalent to their sum in a single long exposure. This is, of course, subject to the general qualification that reparative processes are steadily going on, tending rather gradually to the healing of injured tissue. An ordinary case of photophthalmia completely disappears in less than a week and repair is going on all through this period. It is obviously possible also that apparent complete recovery may still leave the tissues slightly hypersensitive to further injury. We therefore set about investigating the effects of repeated exposures, both liminal and subliminal, to ascertain the additive effect of short exposures, the rate at which the reparative processes proceeded, the completeness of their work, and the possible effects of secondary reactions incidental to the main pathological effects. There was a bare possibility that something akin to anaphylaxis might occur owing to the development of toxins, and this phase of the matter had also to be investigated.

In the case of abiotic radiation affecting a large portion of the body it seems possible that a general constitutional effect might occur owing to the absorption of the toxic substances produced. Such an effect is known to occur after severe burns from heat. In the case of the eye, however, the amount of tissue affected is of course too slight for any such effect to be expected.

The experiments on subliminal exposures repeated at intervals of a few minutes to an hour or more, here summarized, show clearly that within 24 hours the energy effects are simply additive, intermissions within this time evidently being too short for reparative action to take place. The discovery of this fact is important since it shows that with any source of abiotic rays it is the total exposure that counts, and that the effect of this total exposure, if within 24 hours, can be calculated from the data already given.

Experiments.

Effect of repeated exposures. Quartz mercury vapor lamp distance .5 meter.

Subliminal Exposures Repeated within 24 Hours.

Experiment 23. Right eye. Exposure $3\frac{3}{4}$ minutes. Interval 10 minutes. Exposure $3\frac{3}{4}$ minutes. Left eye. Exposed $7\frac{1}{2}$ minutes continuously. Result: Reaction in both eyes, more marked in right.

Experiment 24. Right eye. Exposed five minutes with four intervals of one minute each. Left eye. Exposed 5 minutes continuously. Result: Very slight reaction in each eye.

Experiment 25. Right eye. Exposed 3 minutes. One hour interval. Exposed 3 minutes. Result: Marked reaction.

Experiment 26. Right eye exposed 3 minutes. Four hours interval. Exposed 3 minutes. Result: Marked reaction.

The next phase of the investigation dealt with subliminal exposures at intervals of one or more days, such as might occur in actual use of sources rich in abiotic radiation. The results show that an exposure of one-sixth the liminal repeated every 24 hours for 52 days has no visible effect on the cornea or conjunctiva. An exposure of one-third the liminal repeated every 48 hours has a slight effect on the cornea after seven to nine exposures, which however gradually disappears in spite of the exposures being continued. A daily exposure of one-third the liminal begins to produce a reaction after six exposures. The conjunctivitis disappears, but the corneal effect gradually increases until after thirty-four exposures there is a marked central haze. This leaves a slight corneal scar which is barely visible forty days after the last exposure. An exposure one-half the liminal repeated at the end of 24 hours produces the effect of a single liminal exposure. A single exposure just subliminal, increases the sensitiveness of the eye to abiotic radiation for over two weeks. On the other hand an exposure one-sixth the liminal every 24 hours, or one-third the liminal every 48 hours repeated for a long period of time, has the effect of rendering the eye somewhat less sensitive to abiotic action.

Experiments.

Subliminal Exposures Repeated after 24 Hours. Quartz Mercury Vapor Lamp. Distance .5 Meter.

Experiment 27. Right eye. Exposed 1 minute every day except Sunday for 52 days. (44 exposures.) Result: No reaction throughout the experiment. Three days after last exposure; each eye exposed six minutes. Result: Moderate reaction in each eye, but greater in the left eye.

Experiment 28. Left eye. Exposed 2 minutes every other day, except when Sunday intervened. Results: After 7 exposures, slight stippling of corneal surface. After 9 exposures, slight haze of cornea. After 11 exposures, haze of cornea gone. After 28 exposures, cornea clear, no stippling. After 33 exposures cornea clear, exposures discontinued. Nine days later, each eye exposed six minutes. Results: Right eye, marked reaction. Left eye, much less reaction.

Experiment 29. Left eye. Exposed 2 minutes every day except Sunday. A speculum was used at first but caused ectropion of the lid and was soon dispensed with. Results: No reaction until sixth exposure when there was slight conjunctivitis and stippling of the cornea. After 14 exposures the conjunctival reaction had disappeared but the cornea was distinctly hazy in the centre. After 34 exposures the central haze of the cornea was marked and the epithelial surface showed a number of fine irregular ridges. Exposures discontinued. Forty days later only a barely visible opacity of the cornea remained. Enucleation. Microscopic examination shows Bowman's membrane absent in places, and proliferation and irregular arrangement of the superficial corneal corpuscles.

Experiment 30. Left eye exposed 3 minutes. After 24 hours, no reaction. Left eye exposed 3 minutes. Right eye exposed 6 minutes. Results: Reactions equal in two eyes.

Experiment 31. Right eye exposed 5 minutes. Result: No reaction. Two weeks later. Right eye exposed 4 minutes. Result: Slight reaction.

The next series of experiments had to do with the effect of previous reactions upon the sensitiveness of the eye to subsequent exposures. It was found that previous reactions rendered the eye more sensitive for at least one month, thus reducing the time of exposure necessary

for a liminal reaction. It was found also that if an exposure sufficient to produce a slight reaction, was followed within 24 hours by a subliminal exposure, the total effect was considerably greater than that produced in the control eye by a continuous exposure of the same total length.

Experiments.

Effect of previous reactions upon sensitiveness of eye to subsequent exposures. Quartz mercury vapor lamp.

Experiment 32. Distance .5 meter. Albino rabbit. Right eye exposed 4 minutes. After 24 hours, slight reaction (animal unusually sensitive). Right eye exposed 2 minutes. Left eye exposed 6 minutes. Results: Right eye, increased reaction with loss of corneal epithelium. Left eye, moderate reaction without loss of corneal epithelium.

Experiment 33. Distance .5 meter. Right eye exposed 5 minutes. Result: Very slight reaction. One month later. Right eye exposed $3\frac{1}{2}$ minutes. Result: Moderate conjunctivitis, marked stippling of cornea.

Experiment 34. Right eye exposed 5 minutes at .5 meter. Left eye exposed $7\frac{1}{2}$ minutes at .5 meter. Results: No reaction in either eye. 14 days later. Right eye exposed forty minutes at 35 cm. through crown screen. Left eye exposed 4 minutes at 35 cm. without screen. Results: Slight reactions, more marked in left eye. 8 days later. Right eye exposed 3 minutes at .5 meter. Left eye exposed 4 minutes at .5 meter. Results: Slight reaction in each eye. More marked in right.

Experiment 35. Distance .5 meter. Left eye exposed $7\frac{1}{2}$ minutes. Result: Reaction. 26 days later. Left eye exposed 4 minutes. Result: Slight reaction.

Experiment 36. Right eye exposed $1\frac{1}{2}$ hours at 20 cm. through crown screen. Result: Marked reaction with keratitis, lasting over 2 weeks. 5 weeks later. Left eye exposed 2 minutes at .5 meter. Result: No reaction.

Determination of the Limit of Abiotic Action with Respect to Wave Length.

The critical wave length at which abiotic action on tissue cells ceases has not hitherto been accurately determined. For bacteria it has been found to be about wave length 295 $\mu\mu$. In this connection the observations of Henri [163] and his wife are important. These observers determined the coefficient of absorption of egg albumin for various wave lengths and found that the results corresponded closely

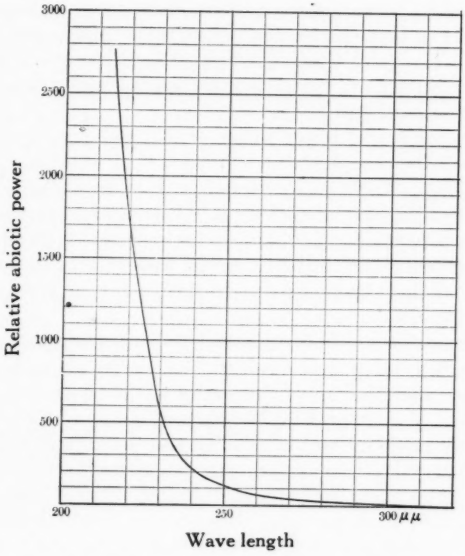


Figure 3. Variation of abiotic power with wave length. (Plotted from Henri's results).

with the time value of bactericidal action for the wave lengths tested. The curve of absorption plotted from their results (Fig. 3) shows that the abiotic action of light with reference to wave length may be expected to diminish rapidly and terminate at about 310 $\mu\mu$. The experiments of Widmark, Hess, and Martin, in which the lens epi-

thelium was injured by exposures through the cornea, prove conclusively that 295 $\mu\mu$ is not the limit for human cells, since the cornea obstructs all waves less than 295 $\mu\mu$ in length. It has frequently been assumed that there is no actual limit of abiotic action but that the latter exists in a diminishing degree through the entire spectrum. Theoretically this may be true, but practically it is not, as our experiments show, and for the following two reasons, namely, first, that in the case of the longer waves and moderate light intensities the abiotic action is so slight as to be readily overcome by the physiological activities of the cells, and second, that in the case of the longer waves and intensities theoretically sufficient to produce abiotic effects the cells are destroyed by heat action, so that there is no opportunity for abiotic effects to become manifest. The real problem may therefore be stated to be the determination of the critical wave length for abiotic action with light intensities just below those sufficient to produce injurious heat effects.

For the experimental investigation of this problem the cornea and lens are of all the tissues of the body the most suitable. This is so because, owing to their great transparency, extreme light intensities are required to produce heat effects in them sufficient to mask abiotic effects. The conjunctiva and skin are far less suitable for this purpose because when the limit of abiotic action with respect to wave length is approached the hyperemia due to heat action overshadows that due to abiotic action.

In this investigation it was necessary to abandon the use of the quartz mercury lamp employed in the earlier experiments. As already noted the spectrum of this source has conspicuous gaps in the very region to be examined for the purpose in hand. It has no lines of perceptible intensity between 334 $\mu\mu$ and the strong double line at 313 $\mu\mu$. Then there is a further gap extending down to the group having its center about 302.5 $\mu\mu$ and another between this group and 297 $\mu\mu$. In fact there are only three rather widely separated lines in this entire debatable region within which the limit sought was known to lie. We therefore turned to the commercial magnetite arc as the most convenient available source since this had already been found by one of us to be particularly rich in the extreme ultra violet. This lamp uses as active electrode an iron tube carrying a compressed mixture of magnetite and of titanium oxide, in a proportion of about 3 of the former to 1 of the latter, opposed to a copper positive electrode. The light is practically all derived from the arc stream produced by the magnetite electrode. The spectrum of this

source is enormously rich in lines due to a complex mixture of those belonging to iron and titanium, reaching down to waves below 230 $\mu\mu$. Moreover, the spectrum is particularly rich in lines between 330 $\mu\mu$ and 290 $\mu\mu$. Beyond this the intensity falls off noticeably. The source is thus particularly well adapted for work in the region here investigated. The lamp used took approximately 9 amperes at the arc which consumed approximately 750 watts. The energy in the ultra violet from wave length 390 $\mu\mu$, was about 15,000 ergs per second per square cm. at .5 meter standard distance, of which approximately 3500 ergs was below 300 $\mu\mu$, as against very nearly 5700 ergs per second per square cm. for the quartz lamp in the same region. The latter source, however, as just pointed out has relatively more energy in the shorter wave lengths. Plate 5 shows side by side the spectra of the two sources in the ultra violet.

For determining the wave length at which abiotic effects on the cornea and lens cease, the use of suitable screens is very much preferable to attempts at using the spectrum formed by a quartz prism as the source of energy. This is for the reason that with screens one can obtain an enormously greater amount of energy than it is practicable to get by passing the radiation through a slit, collimating lens and prism, especially in cases where a considerable area like that of the cornea must be covered. In our experiments seven screens were employed of which the absorption was definitely ascertained by the spectrograph. These screens were of various sorts of optical glass and mostly in the form of discs 43 mm. in diameter and 2 mm. thick. They were as follows:—

		Limits of absorption
1.	Extra dense flint N_D 1.69	335 $\mu\mu$
2.	Medium flint N_D 1.62	315 $\mu\mu$
3.	Medium flint N_D 1.616, 1 mm. thick.	310 $\mu\mu$
4.	Light flint N_D 1.57	305 $\mu\mu$
5.	Medium crown N_D approximately 1.52	300 $\mu\mu$
6.	Extra light flint N_D 1.54	298 $\mu\mu$
7.	Light crown N_D 1.51	295 $\mu\mu$

The absorption of these seven glasses for the magnetite spectrum is shown in Plate 6 with the scale of wave lengths subjoined. These limits are taken at the point where the transmission somewhat abruptly ceases. They do not run to the last lines of which traces are visible, since these are so immensely reduced by the absorption as to have little if any effect bearing on the experiments. In any case the

error would be in the direction of safety, that is, it would tend to set the limit of abiotic action at too long a wave length. It will be noted that in this series of screens the transmission grades off with considerable regularity.

In addition to these screens the following absorbing media were used in some of the experiments. These did not prove of value in determining the limits of abiotic action, but are described here because the experiments in which they were used are important in reference to possible retinal effects, owing to the long exposures given. (Plate 5.)

	Material	Limit of Absorption
8.	$CuCl_2$ 1.5% solution in 5 cm. quartz cell	320 $\mu\mu$, beyond 700 $\mu\mu$
9.	Blue Uviol glass 2 mm.	285 $\mu\mu$ and beyond 470 $\mu\mu$
10.	Auramine O .001% solution in 5 cm. quartz cell	250 $\mu\mu$, 400 $\mu\mu$ to 450 $\mu\mu$
11.	(9 + 10)	

Auramine O, which we tried in several concentrations, is remarkable for the freedom with which it transmits the extreme ultra violet while absorbing the violet end of the visible spectrum rather strongly.

For producing intensive exposures, and particularly for work on the retina the magnetite arc here described was reenforced by the use of a quartz lens system. For one set of experiments we employed two plano convex quartz lenses each of 42 mm. diameter and 18 cm. focal length. These two were generally employed placed with the plane faces in contact either with each other or with one of our screens, making in fact a single lens of 9 cm. focal length for parallel rays. This lens was placed 20 cm. from the arc, an image of which was formed 14 cm. beyond it, at which point the eye was placed. In this set of experiments in addition to abiotic effects in the cornea and lens, small circumscribed heat effects were obtained in the retina analogous to those of eclipse blindness. These will be discussed later (page 697). In another set of experiments the apparatus was assembled as shown in Figure 4. The lenses referred to, B, were placed at 12 cm. from the arc flame A. In the converging cone of rays produced by B, was placed at a distance of 12 cm. therefrom a double convex lens C of quartz cut across the axis, 23 mm. in diameter and of 14 mm. focal length for parallel rays. In the combination used, C brought the rays to a focus at about 12 mm. from its outer apex at or near which point the cornea of the eye D was located in the experiments. The

path of the rays is shown by the dotted lines in the figure. The effect of the arrangement was to pass through the cornea a strongly diverging pencil producing a circle of intense light on the retina. The axial length of the ordinary rabbit's eye is about 16.5 mm. and the ordinary diameter of the area of intense illumination produced by our apparatus was about 11 mm. at the retina as was determined by actual experiment upon a freshly removed eye. With this apparatus a large amount of energy could be concentrated on any required area at or within the surface of the cornea or lens, and by aid of these lens systems we were able to obtain exposures enormously more severe than could possibly be obtained from artificial light sources in ordinary use or than have ever been obtained by previous experimenters in this field. The image was kept fixed during the exposure by slight shifting of the source or lens, since the arc itself tends to wander.

By the use of photo paper the following data were obtained concerning the relative intensities of radiation at the focus and on the retina with the double lens system. The size of the area of most

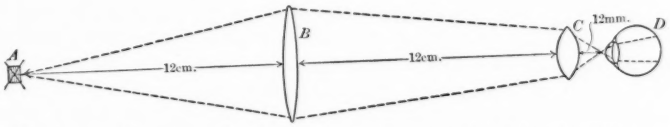


FIGURE 4. Quartz condensing system. Screens omitted for simplicity.

intense illumination at the focus was $2 \times 4\frac{1}{4}$ mm. The diameter of effective illumination at a distance of 16 mm. from the focus, corresponding to the position of the retina, was 11 mm. Through a euphos glass screen an exposure of 5 seconds at the focus closely corresponded in intensity to an exposure of 75 seconds at the position of the retina, so that neglecting absorption by the media of the eye the intensity of the illumination of the retina with this lens system was about fifteen times less than that of the cornea. This ratio was confirmed by photographs taken with a model schematic eye without screen and with a picric acid screen, so that it may be assumed to hold over a very wide range of wave lengths.

The results of these experiments are given below. For purposes of comparison other experiments with the magnetite are also given here, although they have no direct bearing on the determination of the critical wave length of abiotic action. For the same reason experiments are given showing the length of exposure to the quartz mercury

vapor lamp through a crown screen (295 $\mu\mu$) necessary to produce photophthalmia. The thermic effects produced in these experiments as well as the character of the abiotic effects are discussed elsewhere (pages 662 and 692).

For determining the critical wave length of abiotic action it will be seen that the crucial experiments were Experiments 81 to 85. These showed that for light of extreme intensity no effects either abiotic or thermic, were produced on the cornea or lens epithelium by an exposure of $1\frac{1}{2}$ hours to waves over 315 $\mu\mu$ in length, and that no abiotic effects but slight thermic effects were produced by light containing wave lengths of 310 $\mu\mu$ and longer. Light containing wave lengths of 305 $\mu\mu$ and longer, produced marked thermic effects but no abiotic effects after one hour exposure, but after $1\frac{1}{2}$ hours exposure produce marked thermic effects and the slightest possible trace of abiotic effects. That is to say, the limit of abiotic action with reference to wave length for corneal cells is almost exactly 305 $\mu\mu$. In Experiment 82, in which the light was focussed on the cornea, the only evidence of abiotic action was the loss of corneal epithelium, while in the Experiment 83, in which the light was focussed on the anterior surface of the lens, the only evidences of such action were the slight but characteristic changes in the lens epithelium. In any other available tissues than the cornea and lens such slight abiotic effects would undoubtedly have been completely masked by extreme heat effects, but in none of these experiments did the lens show the slightest thermic effects.

The insignificance of the abiotic action at wave length 305 $\mu\mu$ becomes more apparent when the equivalent critical time is computed for exposure to the direct radiations from the magnetite arc. Our experiments show that the intensity of light at the focus of the double lens system is at least eighteen times the intensity of the bare arc at a distance of 20 cm. This means that to obtain slight loss of corneal epithelium with direct waves of 305 $\mu\mu$ in length from the magnetite arc, an exposure of 27 hours at a distance of 20 cm. or an exposure of 28 days at a distance of 1 meter would be required. To produce mild photophthalmia the time required would be about one-third these figures. As a matter of fact however, our experiments on frequently repeated subliminal exposures, already given, prove that at 1 meter no effects whatever would be produced by such slight abiotic action per unit of time owing to the vital activities of the cells.

Similar calculations for the portion of the spectrum including only waves longer than 295 $\mu\mu$, as well as direct experiments, show that

an exposure of 20 minutes at 20 cm., in case of the magnetite arc, is required to produce photophthalmia, or an exposure of $8\frac{1}{3}$ hours at 1 meter. The ratio of the abiotic activity of the whole spectrum to that of the portion exceeding 295 $\mu\mu$ is $\frac{18}{1}$. This holds approximately true also for the quartz mercury vapor lamp.

In regard to the lens epithelium, it should be noted that only abiotic changes were obtained, and these with wave lengths as long as 305 $\mu\mu$ as already stated. With light of this wave length, however, as shown by the above computation it is evident that they could not be obtained by means of the direct light from any artificial light sources at any distance at which the eye could bear the heat. With wave lengths of 295 $\mu\mu$ and over, the lens epithelium was affected by an exposure of 5 minutes to the double lens system, the liminal exposure probably being about 3 minutes. Since the cornea itself cuts off the waves at this point, it follows that an exposure to the bare magnetite arc of 54 minutes at 20 cm. or of 22 hours at 1 meter would be required to affect the lens epithelium. Hess, strangely enough, was unable to obtain lens changes through a screen transparent to waves of 280 $\mu\mu$, and drew the inconsistent conclusion, in view of the fact that the cornea was known by him to obstruct still longer waves, that lens changes are produced only by the very short waves of the spectrum. The character of the lens changes produced in our experiments is described on page 671.

Judging by the effects on the cornea, the abiotic intensity at the focus for the single lens system was about $\frac{1}{3}$ that for the double lens system.

Comparing the results obtained with the quartz mercury vapor lamp and those with the magnetite arc it is found that the abiotic activity of the entire spectrum of each is in about the ratio of 6 for the mercury lamp to 5 for the magnetite arc. This ratio holds with and without the crown screen (295 $\mu\mu$).

These ratios obtained from the pathological effects are in fairly close accord with those derived from the experiments of one of us by purely radiometric methods. Taking average conditions of the two sources here referred to, the abiotic radiations from the quartz lamp should aggregate about 4200 ergs per second per square cm. at the standard distance of .5 meter. The magnetite arc as used gave abiotic radiations aggregating about 3300 ergs per second per square cm. at the same distance. The ratio between these two quantities is 5 to 6.35 as compared with the 5 to 6 of the pathological results, an agreement quite as close as could reasonably be expected considering the nature of the case.

Full grown rabbits were used in all the experiments and the light was focussed upon the cornea unless otherwise stated. In all experiments relating to the retina the pupil was previously dilated by a mydriatic. In the experiments with the double lens system the conjunctiva was not directly exposed to the light so that the conjunctivitis noted was largely secondary. The iris however was sometimes more or less exposed.

Experiments.

Quartz mercury vapor lamp. Crown glass screen (295 $\mu\mu$).

Distance 35 cm.

Experiment 37. Exposed 20 minutes. No reaction.

Experiment 38. Exposed 40 minutes. Little if any reaction.

Experiment 39. Exposed 40 minutes. Little if any reaction.

Experiment 40. Exposed 60 minutes. Moderate conjunctivitis. Cornea clear. Iris congested.

Distance 20 cm.

Experiment 41. Exposed 1½ hours. Marked purulent conjunctivitis. Cornea very hazy in central area. Iris congested and hemorrhagic. The conjunctivitis persisted about 10 days. The corneal haze and a few minute iris hemorrhages were visible over 4 weeks.

Magnetite arc. No lenses or screens.

Experiment 42. Distance 50 cm. Exposed 6 minutes. Slight conjunctivitis. Stippling of corneal surface without loss of epithelium. (Liminal reaction.)

Experiment 43. Distance 20 cm. Exposed 1 minute. Slight conjunctivitis. Corneal epithelium intact.

Experiment 44. Exposed 2½ minutes. Moderate purulent conjunctivitis. Cornea stippled but epithelium intact.

Experiment 45. Exposed 4 minutes. Marked conjunctivitis with edema and punctate hemorrhages. Cornea hazy and epithelium lost from ¾ of its surface. Microscopic examination (48 hrs.) shows leucocytic infiltration of cornea, but corpuscles and endothelium normal. Lens epithelium normal.

Experiment 46. Exposed 15 minutes. Marked conjunctivitis. Haze of cornea with loss of epithelium. Microscopic examination

(48 hrs.); shows leucocytic infiltration of cornea. Corpuscles slightly affected in outermost layers. Endothelium normal. Lens epithelium normal. Iris normal. Serum in anterior chamber.

Experiment 47. Exposed 16 minutes. Result same as in previous experiment.

MAGNETITE ARC. CROWN GLASS SCREEN (295 $\mu\mu$).

Experiment 48. Distance 20 cm. Exposed 20 minutes. Slight conjunctivitis. Cornea clear, not stippled, epithelium intact.

Experiment 49. Distance 20 cm. Exposed 40 minutes. Well marked conjunctivitis with edema and slight purulent discharge. Cornea clear. Reflex impaired, but epithelium intact — does not stain.

Experiment 50. Water cell. Distance 14 cm. Exposed 22 minutes. Moderate conjunctivitis. Corneal epithelium intact. Microscopic examination (48 hrs.). Cornea, iris, and lens epithelium normal. No serum in anterior chamber.

MAGNETITE ARC. QUARTZ SINGLE LENS SYSTEM. WATER CELL.

Experiment 51. Albino. Crown glass screen (295 $\mu\mu$). Exposed 20 minutes. Immediate enucleation. Microscopic examination shows cornea, iris, and lens epithelium normal.

Experiment 52. Pigmented eye. Crown glass screen (295 $\mu\mu$). Exposed 6 minutes. Moderate conjunctivitis. Slight haze of cornea without loss of epithelium. Microscopic examination (4 days): Corneal stroma shows slight leucocytic infiltration. Corpuscles and endothelium normal. Lens epithelium normal.

Experiment 53. (Pl. 4, Fig. 12). Albino. Crown glass screen (295 $\mu\mu$). Exposed 12 minutes. Purulent conjunctivitis. Cornea hazy, shows loss of epithelium. Iris congested and hemorrhagic. Microscopic examination (48 hrs.): Corneal corpuscles show marked abiotic changes. Endothelium absent. Iris shows interstitial hemorrhages. Lens epithelium shows moderate abiotic changes. Retina shows burned spot.

Experiment 54. (Pl. 3, Fig. 9). Albino. Crown glass screen (295 $\mu\mu$). Exposed 20 minutes. Marked reaction with loss of corneal epithelium. Microscopic examination (2 days): Most of the corneal corpuscles destroyed in central area, endothelium absent. Marked abiotic changes in lens epithelium. Iris shows interstitial hemorrhages.

Experiment 55. (Pl. 2, Fig. 5). Albino. Crown glass screen (295 $\mu\mu$). Exposed 1 hour. Marked reaction with loss of epithelium. Microscopic examination (24 hrs.): Corneal corpuscles destroyed in central area, endothelium absent, marked abiotic changes in lens epithelium. Iris shows interstitial hemorrhages and slight purulent exudation from vessels. Retina shows burned area.

Experiment 56. Pigmented eye. No screen. Exposed 20 minutes. Marked reaction with loss ($\frac{3}{4}$) of corneal epithelium. Beginning vascularization of cornea on 6th day. Microscopic examination (6 days): Corneal epithelium reformed, corpuscles largely destroyed, endothelium absent. Lens epithelium shows marked changes. Iris: Most of the stroma cells destroyed in anterior half of iris for a distance of 1.5 mm. from the pupillary margin. Some of the stroma cells show characteristic granules. A few mitotic figures seen. Endothelium entirely absent from some of the blood vessels. Interstitial hemorrhages.

Experiment 57. Albino. Crown glass screen (300 $\mu\mu$). Exposed 15 minutes. Little if any reaction. Microscopic examination (6 days): Cornea, iris, and lens epithelium unaffected. Retina shows heat effect involving pigment epithelium only.

Experiment 58. Pigmented eye. Flint glass screen (315 $\mu\mu$). Exposed 1 hour. No reaction. Microscopic examination (6 days): Cornea, iris, and lens epithelium unaffected. Retina shows burned spot.

Experiment 59. Albino. Flint screen (335 $\mu\mu$). Exposed $1\frac{1}{2}$ hours with 5 minutes intermission at end of 30 minutes. No reaction. Microscopic examination (2 days): Cornea, iris, and lens epithelium unaffected. Retina and chorioid show burned area.

MAGNETITE ARC. QUARTZ DOUBLE LENS SYSTEM. WATER CELL. NO SCREEN.

Experiment 60. Exposed 5 seconds. No effect.

Experiment 61. Exposed 10 seconds. Slight reaction. Corneal epithelium lost in exposed area.

Experiment 62. Exposed 10 seconds. Result same as in Experiment 61.

Experiment 63. Exposed 30 seconds. Marked reaction. Slight haze of cornea with loss of epithelium.

Experiment 64. Exposed 5 minutes. Marked reaction. Softening of corneal stroma. Translucent corneal scar at end of two months.

In the following experiments (see page 668) the exposure was sufficient to produce marked photophthalmia with loss of corneal epithelium and endothelium, destruction of corneal corpuscles, softening and swelling of the stroma, and marked changes in the lens epithelium. The retina was normal in all.

Experiment 65. Exposed 6 minutes. Enucleation at end of 4 days.

Experiment 66. Exposed 20 minutes. Enucleation at end of 10 hours.

Experiment 67. (Pl. 2, Fig. 6). Exposed 20 minutes. Enucleation at end of 48 hours.

Experiment 68. Exposed 20 minutes. Enucleation at end of 4 days.

Experiment 69. (Pl. 1, Fig. 1). Exposed 20 minutes. Enucleation at end of 12 days.

Experiment 70. (Pl. 3, Fig. 7). Exposed 20 minutes. Enucleation at end of 2 months.

MAGNETITE ARC. QUARTZ DOUBLE LENS SYSTEM. WATER CELL.

With crown screen (295 $\mu\mu$):

Experiment 71. Exposed 2 minutes. Slight conjunctival reaction. Corneal reflex impaired but epithelium intact.

Experiment 72. Exposed 3 minutes. Loss of corneal epithelium in exposed area.

In the following experiments there was marked keratitis and abiotic changes in the lens epithelium. The retina was normal.

Experiment 73. Exposed 5 minutes. Enucleation at end of 48 hours.

Experiment 74. Exposed 20 minutes. Enucleation at end of 5 days.

Experiment 75. Exposed 20 minutes. Enucleation at end of 10 days.

Experiment 76. Exposed 20 minutes. Enucleation at end of 34 days.

With .001 auramine O solution in quartz cell 5 cm. thick (substituted for water cell), *and blue uviol screen,* the two together obstructing all waves less than 250 $\mu\mu$ and longer than 470 $\mu\mu$ in length (see Pl. 5).

Experiment 77. Exposed 45 minutes. Enucleation at end of 3

days. Marked keratitis and abiotic changes in lens epithelium. Retina normal.

With flint glass screen (298 $\mu\mu$):

Experiment 78. (Pl. 4, Fig. 11). Exposed 1 hour. After 20 minutes: Cornea shows well marked central haze (heat effect). Epithelium intact. After 24 hours: Slight conjunctival reaction. Haze of cornea greater. Central loss of epithelium (5 mm.). After 72 hours: Corneal epithelium reformed. Haze of stroma persists. Enucleation. Eye immediately opened. Fundus bisected vertically. One half fixed in saturated solution of mercuric chloride. The other stained by the vital methylene blue method. Lens fixed in Zenker's fluid. Microscopic examination: Corneal stroma swollen to $\frac{7}{5}$ normal thickness. Corneal corpuscles completely destroyed in posterior two-thirds, present, but show characteristic abiotic changes in anterior third. Endothelium necrotic and absent in places. Lens epithelium shows marked abiotic changes. Retina normal.

With crown glass screen (300 $\mu\mu$):

Experiment 79. Exposed 1 hour. Slight conjunctival reaction. Marked central haze of cornea with loss of epithelium. Microscopic examination (48 hrs.): Corneal stroma swollen to twice normal thickness. Epithelium, corneal corpuscles, and endothelium completely destroyed in central area. Towards the periphery corpuscles first show abiotic changes then proliferative changes (heat effect). Lens epithelium shows slight but definite abiotic changes — swelling of cells and characteristic granules. Iris normal (unexposed). Retina normal.

Experiment 80. Exposed 20 minutes. Slight central haze of cornea persisting over 9 days. Epithelium intact.

With flint glass screen (305 $\mu\mu$):

Experiment 81. (Pl. 1, Fig. 2). Exposed 1 hour. Immediately after exposure; cornea perfectly clear. After one hour: Cornea shows distinct central haze. After 24 hours: Cornea more hazy, epithelium intact and normal. After 3 days: Haze of cornea persists. There has been no loss of epithelium on daily examination. Microscopic examination (3 days): Corneal epithelium intact. Stroma swollen to $\frac{5}{3}$ normal thickness. Endothelium absent. Corpuscles invisible in posterior $\frac{1}{4}$ of exposed area, present and actively proliferating in anterior portion. No evidences of abiotic action. (Iris not exposed). Lens epithelium normal. Retina normal.

Experiment 82. Exposed $1\frac{1}{2}$ hours. Immediately after exposure: cornea shows faint haze. After 45 minutes: Corneal haze more

distinct. After 1 hour: Haze more distinct. Epithelium intact. After 2 hours. Epithelium intact. Slight diffuse deep staining of stroma with fluorescine. Animal avoids obstacles by sight (other eye absent). After 24 hours: Cornea hazy, shows loss of epithelium (3 mm.). After 48 hours: Epithelium reformed. Microscopic examination (48 hrs.); Corneal epithelium intact but thin. Stroma swollen to almost twice normal thickness. Corpuscles completely destroyed in posterior portion of exposed area, a few present in anterior portion show no abiotic changes. At periphery of exposed area the cells are actively proliferating. Endothelium absent. (Iris not exposed). Lens epithelium normal. Retina normal.

Experiment 83. Pigmented eye. Light focussed on anterior pole of lens instead of on cornea as in previous experiment. Pupil not fully dilated. Total exposure $1\frac{1}{2}$ hours. Exposed 30 minutes. During the exposure, the pupil became contracted. Immediately after exposure: Cornea clear. After 50 minutes: Cornea shows distinct haze. After 1 hour: Exposed 1 hour. Immediately after exposure: Iris congested, pupil contracted (about $1\frac{1}{2}$ mm.) and irregular, does not dilate in the dark. Cornea more hazy. After 2 hours. Corneal haze very marked. Epithelium intact, does not stain. Pupil slightly larger. After 24 hours: No conjunctival reaction. Pupil dilated, but not fully so. Cornea shows marked haze without loss of epithelium. After 48 hours: Conditions about the same. Epithelium intact, does not stain. Microscopic examination: (48 hrs.): Corneal epithelium intact. Stroma swollen to $\frac{5}{3}$ normal thickness. and corpuscles affected as in previous experiment. Endothelium absent. Iris normal. Retina normal. Lens epithelium shows slight but definite abiotic changes. Cells are slightly swollen in exposed area and a few of them contain characteristic granules.

With flint glass screen (310 $\mu\mu$):

Experiment 84. Exposed $1\frac{1}{2}$ hours. After 24 hours: Marked central haze of cornea. Epithelium intact, does not stain. After 2 days: Haze of cornea about the same. Epithelium intact, does not stain. After 4 days: Cornea clearer. Epithelium intact. Microscopic examination (4 days): Epithelium intact. Corneal stroma swollen to about $\frac{4}{3}$ normal thickness. Corneal corpuscles nowhere destroyed, show marked proliferative changes, especially in posterior portion of cornea. No abiotic changes seen. Endothelium absent in places. Lens epithelium normal. Retina normal.

With flint glass screen (315 $\mu\mu$):

Experiment 85. Exposed $1\frac{2}{3}$ hours. After 24–48 hours: No re-

action. Cornea clear. Microscopic examination (48 hrs.): Cornea, lens epithelium, and retina normal.

With 1.5 % copper chloride solution substituted for water in quartz cell, crown glass screen (295 μμ) and blue uviol screen, the whole obstructing all waves less than 320 μμ and longer than 700 μμ in length.

Experiment 86. Exposed 1 hour. No effect. Microscopic examination (48 hrs.): Cornea, lens epithelium and retina normal.

With 1.5 % copper chloride solution and blue uviol screen, obstructing all waves less than 320 μμ and longer than 700 μμ in length:

Experiment 87. Exposed 1 hour. No effect. Microscopic examination (3 days): Cornea, lens epithelium, and retina, normal.

With flint glass screen (315 μμ). (The water cell leaked so that for an unknown length of time the eye received also infra red rays):

Experiment 88. (Pl. 1, Fig. 3). Albino. Exposed 1 hour. After 24 hours: No conjunctivitis. Marked haze of cornea, epithelium intact. After 48 hours: Haze persists. Epithelium intact. Microscopic examination (48 hrs.): Corneal epithelium intact. Stroma swollen to double normal thickness, stains faintly in eosin. Corpuscles and endothelium completely destroyed in exposed area. At periphery of affected area corpuscles show active proliferation with mitoses (page 693). Iris shows a few minute hemorrhages near pupil. Lens epithelium normal. Retina: Pigment epithelium shows distinct heat effect over an area 6 mm. in diameter. The cells are swollen or stain deeply in eosin, and their nuclei are often pycknotic. Otherwise the retina is normal.

With crown glass screen (295 μμ):

Experiment 89. Aphakic eye. Pigmented. Exposed 35 minutes with intermissions of 1 minute every five minutes. Light focussed on opening in lens capsule. After 24–48 hours: Marked keratitis with loss of epithelium. Microscopic examination (48 hrs.): Cornea shows typical abiotic changes. Epithelium and endothelium absent. Iris (only slightly exposed) shows a few minute hemorrhages. Pigment epithelium of retina shows marked heat effect over an area 4 mm. in diameter. Ganglion cells and other retinal elements normal.

With flint glass screen (315 μμ) but without water cell:

Experiment 90. Pigmented eye. Atropine mydriasis. Exposed 30 minutes. Immediately after exposure: Cornea clear. Pupil contracted. After 20 minutes: Distinct haze of cornea. No conjunctivitis. After 4 hours: Haze of cornea marked. Epithelium intact, does not stain. No conjunctivitis. Pupil larger, vertically oval. Prompt lid reflex to light. After 24 hours: No conjunctivitis.

Marked haze of cornea. Epithelium intact. Pupil widely dilated. After 48 hours: Condition about the same. Microscopic examination (48 hrs.): Corneal stroma swollen to $\frac{5}{3}$ normal thickness. Epithelium intact. Corneal corpuscles completely invisible in central portion of exposed area. At the periphery of the latter they show active proliferation many of them being in mitosis. Endothelium absent beneath exposed area. Iris and lens epithelium normal. Retina normal — pigment epithelium unaffected.

With flint glass screen (335 μμ), but without water cell.

Experiment 91. Pigmented eye. Exposed 30 minutes. After 24–48 hours: No reaction, cornea clear. Microscopic examination (48 hrs.): Cornea, lens epithelium, and retina normal. Pigment epithelium of retina normal.

SUNLIGHT. BLUE UVIOL SCREEN AND .001% SOLUTION OF AURAMIN O IN QUARTZ CELL 5 CM. THICK.

Light focussed on cornea by large quartz lens 12 cm. in diameter and 25 cm. in focal length. Atmosphere not perfectly clear (April 2, 1913):

Experiment 92. Albino. Exposed 45 minutes. After 24 hours: Very slight conjunctival reaction. Cornea hazy, epithelium intact. Iris congested. After 48 hours: Condition about the same. Corneal epithelium intact. Microscopic examination (48 hrs.): Corneal epithelium intact. Stroma swollen to about $\frac{4}{3}$ normal thickness. Corpuscles and endothelium completely destroyed in exposed area. At periphery corpuscles show proliferative changes. Affected area wider posteriorly than anteriorly. No characteristic abiotic changes made out. Iris normal. Retina congenitally defective (coloboma of optic disc, ganglion cells few in number) shows no abiotic or heat effects. Pigment epithelium normal.

MAGNETITE ARC. QUARTZ SINGLE LENS. WATER CELL. CROWN GLASS SCREEN (295 μμ).

Experiment 93. Right eye exposed 8 minutes. Left eye exposed 4 minutes. One hour later; Left eye exposed 4 minutes. After twenty-four hours: Slight reaction in each eye without loss of epithelium. After 3 days: No reaction. Magnetite arc. Double lens system. Water cell. Crown screen (295 μμ). Exposed left eye

3 minutes. After 1½ hours: Exposed right eye 6 minutes; left eye. 3 minutes. Marked reaction with haze of cornea and loss of epithelium in each eye. Microscopic examination: (5 days after first exposures): Cornea of each eye shows marked abiotic changes with loss of epithelium and endothelium. Lens epithelium shows abiotic changes in both eyes, but more marked in left eye.

Histological Technique.

Fixation: In a few of the earlier experiments the eyes were fixed in a warm saturated mercuric chloride solution as recommended by Birch-Hirschfeld. This was not found, however, superior to Zenker's fluid for demonstrating the structure of the ganglion cells, particularly the Nissl bodies, and Zenker's fluid at room temperature was therefore used for fixation in all except one of the experiments relating to the retina. Before opening the eye it was usually placed in the fixing fluid for about ten minutes. This prevented the cornea from losing its shape and the sclera and retina from becoming distorted as happened when the eye was immediately opened. The eye was then incised all around at the ora serrata, the vitreous body gently lifted out, the lens removed and the two portions replaced in the Zenker's fluid for four to six hours. Longer fixation gives less brilliant results. After fixation the tissues were washed in running water twenty-four hours.

Embedding: Celloidin embedding was employed in all except one experiment in order to avoid the shrinkage that results from the paraffine process.

Cornea and Iris: Meridional sections 8 to 10 μ in thickness were always made, passing through the middle of the most affected part of the cornea and the centre of the pupil. Tangential sections of the cornea 6 to 8 μ in thickness were also frequently made. The sections were stained in alum hematoxylin followed by .2% solution of water soluble eosin in 80% alcohol.

Lens Capsule: The most satisfactory method of demonstrating changes in the capsular epithelium is by means of flat preparations. This method was used by Hess [179] and later by Martin,[238] but Birch-Hirschfeld [37] speaks of using flat sections. The method as we have carried it out is as follows: The eye is opened as already described, by an incision passing just behind the ciliary body all around. The zonule is then cut all around by means of scissors, care being taken

not to rupture the capsule, and the lens removed and placed in Zenker's fluid for two hours. The lens may be fixed in situ and removed afterwards, but this causes the iris epithelium to adhere to it. Birch-Hirschfeld mistakenly regarded such adhesions, which he found in the exposed eyes, as pathological. They may be removed by gently rubbing the capsule with wet filter paper. The lens is now rinsed in water and the capsule incised all around the equator with a sharp knife. The anterior capsule is now readily stripped off, floated in water and treated as follows: Lugol's solution (1%) a few seconds. Water. 95% Alcohol two minutes or longer. Water. 10% aqueous solution sodium hyposulphite until color of iodine is removed. Water. The capsule will be found to curl toward the cell free side. It is now floated upon a piece of paper and by means of scissors five radial incisions are made through both paper and capsule reaching to within a short distance of the centre. It is then freed from the paper and floated upon a cover glass with the curled edges up, so that the epithelium is in contact with the glass and thus will be nearest the lens of the microscope. The curled edges are flattened out by stroking with bits of filter paper, which removes the excess of water and prevents the edges curling again. The preparation is now blotted firmly with filter paper. Alum hematoxylin until deeply stained. It is best to use a sharply acting hematoxylin solution and avoid differentiating in acid alcohol as the latter is apt to act unevenly. Water. 0.2% solution of water soluble eosin in 80% alcohol, 30 minutes. Water. The preparation is now thoroughly dehydrated in absolute alcohol, cleared in oil of origanum followed by xylol, blotted again if necessary, and mounted on a slide in xylol-balsam.

Retina: Vertical sections of the retina 6 μ to 8 μ in thickness, were made in all cases. These always included the optic disc and the area below it that had been exposed to the light during the experiment. This area contains a much larger proportion of ganglion cells than any other part of the retina and may be regarded as analogous to the human macula, although it is much larger and less sharply defined. The ganglion cells are similar to those of the human macula, but never occur in more than a single row. Plane sections of the retina were also often made, and these were found to give the best demonstration of the ganglion cells.

Sections were always stained in eosin and thionin, which is probably the most satisfactory method for demonstrating Nissl bodies and at the same time gives a beautiful general stain of the retina. Dilute aqueous solutions of thionin rapidly lose in staining power, so that it is important that they be always freshly prepared. The fol-

lowing carbol-thionin solution devised by one of us retains its properties indefinitely and from it a powerful staining solution may be made at once by the simple addition of water:

Thionin to saturation, about	.3 gm.
Absolute alcohol,	60 cc.
Phenol crystals (melted)	30 cc.

For use, add one full drop of this solution to 2 cc. of distilled water. Sections are stained as follows:

(1) Lugol's solution 1:2:100, 1 minute, followed by water, 95% alcohol and sodium hyposulphite solution, to remove mercurial precipitates. Water.
(2) 0.2% solution of water soluble eosin in 80% alcohol, 5 minutes. Water.
(3) Carbol thionin diluted immediately before use as above, 5 minutes. Water.
(4) Differentiate and dehydrate in 95% alcohol, two changes, until excess of thionin is removed and sections show well marked eosin stain, about 30 seconds.
(5) Oil of origanum.
(6) Place on slide, blot, wash in xylol, blot, xylol-balsam.

To obtain the most brilliant results it is important not to overstain the sections in thionin solution as it is then impossible to produce sharp differentiation of the Nissl bodies by treating with alcohol. The results also are more brilliant the shorter the time that has elapsed between the fixation of the tissues and the staining of the sections.

The Character of the Reactions of the Ocular Tissues to Abiotic Radiations.

conjunctiva and cornea.

Clinical: Our experiments show that the effects on the conjunctiva and cornea of moderate exposures to waves less than 295 $\mu\mu$ in length do not differ qualitatively in their clinical aspects from those produced by longer exposures to waves from 295 $\mu\mu$ to 305 $\mu\mu$ in length. Severe exposures, however, produce markedly different effects on the cornea in the case of the short waves than in the case of the longer waves, owing to the fact that the latter are not fully absorbed by the corneal stroma. The effects of severe exposures to very short waves is there-

fore not included in the following description, but will be given separate consideration. A description of combined thermic and abiotic effects on the cornea in certain experiments, resulting from prolonged intense exposures is given on page 694.

After exposure of a rabbit's eye to light containing abiotic rays, no immediate changes take place, however great the intensity, provided a heat effect is not produced, and symptoms of irritation do not usually appear for several hours. In other words, there is a latent period before any visible effects are produced. This exists not only as regards clinical symptoms but also as regards histological changes. In a general way it varies inversely as the severity of the exposure, but in no case is the first appearance of symptoms delayed longer than twenty-four hours. That is to say, a latency longer than this corresponds to an exposure too slight to produce any demonstrable effects. The shortest latent period observed by us was thirty minutes. This occurred after intense exposure to the short waves of the magnetite arc, as described later. The least effect that occurs after exposure to abiotic radiation consists in slight hyperemia of the conjunctiva. After more intense exposures the congestion is correspondingly greater and is associated with edema and purulent exudation. There also may be conjunctival ecchymoses. The cornea, after exposures sufficient to produce slight conjunctivitis, remains clear and shows only slight stippling of the surface. After longer exposures the cornea becomes hazy in a rather sharply defined central area. This delimitation is no doubt due chiefly to the fact that the rays strike the periphery of the cornea obliquely so that there is less light here per unit area, and to a less extent to the greater loss by reflection at the periphery (see diagram, page 634). Over the central area the epithelium shows marked stippling and is then cast off, usually, however, not until about 24 hours. The loss of epithelium sometimes cannot be determined without the use of fluorescine staining, owing to the margins of the defect not then being sharply defined. This is due to the fact as shown by microscopic examination, that the epithelium usually becomes thinned by desquamation before solution of continuity occurs.* The haziness of the cornea usually reaches its

* The cornea of a rabbit's normal eye often shows punctate spots and irregular lines after staining with fluorescine that closely resemble the lesions of dendritic keratitis. These are due to defects in the epithelium so small that they do not easily become visible until the stain has diffused through them into the corneal stroma, which requires one or two minutes. They are possibly due to the infrequent winking for which rabbits are noted. They cannot be mistaken by anyone familiar with their appearance for erosions due to exposure to abiotic radiations, because the latter stain almost instantaneously and are much larger and sharply defined.

maximum in about 48 hours, when, as will be pointed out, there is some leucocytic infiltration.

After 3 days the purulent conjunctival discharge becomes less, but it may not entirely subside for about 9 days. The corneal epithelium is usually reformed on about the 4th day. Haziness of the cornea noticeably begins to subside in 3 to 10 days. After five weeks only a slight central haze remains. Following sufficiently intense exposures, new vessels are seen extending into the cornea from the limbus in about six days.

The conjunctival reaction that occurs after moderate exposure to abiotic radiations, is only in very small part reflexly due to irritation of the cornea. This is proved by several experiments in which the cornea was exposed through a diaphragm which protected the conjunctiva. Here, although the cornea was markedly affected, and the epithelium destroyed, the conjunctiva showed no reaction until after about 48 hours, and then only slight hyperemia.

The foregoing description applies to the effect produced on the cornea by moderate exposures to the bare mercury vapor quartz lamp, or bare magnetite arc, and by relatively long exposures (5 to 20 minutes) to the magnetite arc through a water cell, quartz lens system, and crown screen. The latter absorbs all rays less than 295 $\mu\mu$ in length and thus protects the corneal stroma from injury. With the magnetite arc, and quartz lens system, but without any screen, a very much greater as well as different effect may be produced. With this arrangement and an exposure of 20 minutes a dosage is obtained that is more than one hundred and fifty times as great as that of a liminal exposure necessary to produce slight keratitis. Following such an exposure the following changes occur. Immediately after the exposure the cornea is perfectly clear. At the end of thirty minutes there is slight hyperemia of the conjunctiva and central haziness of the cornea. At the end of four hours the conjunctivitis is marked and the corneal haze much greater. The exposed area is completely anaesthetic. The epithelium is intact, but stains slightly in flourescine. The iris is highly congested. At the end of twenty-four hours there is a marked general inflammatory reaction of the conjunctiva with oedema and purulent discharge. The epithelium is lost from the exposed area in twenty-four hours, and reformed about thirty-six hours later. On the fourth or fifth day the cornea, without becoming more hazy, begins to swell in the exposed region. This swelling increases and the affected area becomes softened until an appearance is produced on about the eighth day of a large flaccid

vesicle involving two-thirds the area of the cornea. This condition remains almost unchanged until about the thirteenth day, except that on about the sixth day vascularization of the cornea is observed. On about the fourteenth day the inflammatory reaction, which has almost completely subsided, begins again. This is probably a reaction of repair. During this time the process of vascularization makes rapid progress and the new vessels invade the central area which is now somewhat firmer, but still pits when touched by a probe. On the twenty-fifth day the inflammatory reaction is again almost gone and the new vessels have begun to disappear. The exposed area is now only slightly swollen and no longer pits, but is very cloudy. On the thirty-third day the vessels have largely disappeared. The exposed area is no longer swollen and presents a translucent appearance. After two months the surface of the cornea is smooth and there is a translucent interstitial opacity. The repair of the injury is much more complete than could be expected in the case of a human cornea.

After an exposure of five minutes *i. e.* one-fourth the former dosage, to the magnetite arc through the quartz lens system and water cell the cornea undergoes softening in the exposed area as in the case of the longer exposures. The injury, however, is repaired without vascularization of the cornea, leaving a central translucent scar.

The Histological Changes Produced in the Cornea by Abiotic Radiations.

The histological changes produced in the cornea by abiotic radiations were studied chiefly in eyes exposed to the magnetite arc with and without interposition of quartz lenses and various screens. Corresponding to the differences in the clinical effects, different histological effects were obtained when a crown glass screen was used than when it was omitted. The chief difference was that with the crown screen the corneal stroma escaped injury, due to the fact that it was then protected from all waves which it strongly absorbed, namely, waves less than 295 $\mu\mu$ in length. With the crown screen, exposures sufficient to destroy the epithelium always severely injured the corneal corpuscles. Without the crown screen, on the other hand, owing to the greater abiotic activity of the short rays stopped at the surface of the cornea, the epithelium was destroyed by exposures too short to have any visible effect on the corneal corpuscles. On account of these differences the histological effects produced by the short waves and relatively long waves will be described separately.

The Histological Changes Produced in the Cornea by Abiotic Waves over 295 $\mu\mu$ in Length.

Since, for reasons already given, the central portion of the cornea under the usual conditions of the experiments is much more strongly affected than the periphery, the various degrees of injury produced are easily made out by examining the cornea from the periphery towards the centre. Examined in this way twenty-four to forty-eight hours after exposure, it is found that the epithelium first shows spacing out of its basal cells, and then in addition desquamation of the superficial layers until finally the epithelium is more or less abruptly cast off. At the margins of this erosion the individual epithelium cells show changes similar to those met with in the case of the lens capsule, that is, formation within the cytoplasm of eosinophilic and basophilic granules. Swelling of the cells, however, is not noticeable, possibly because the cells are cast off when this occurs. The nuclei are relatively little affected, although some of them are pycknotic. Mitotic figures are observed only in the apparently normal epithelium at the periphery of the cornea.

After exposures through a crown screen (295 $\mu\mu$) sufficient to produce injury to the lens capsular epithelium, the corneal lamellae show slight if any changes; possibly they stain less deeply in eosin. The corneal corpuscles, however, show marked changes. Just as in case of the lens epithelium, all of the cells are not equally injured and certain cells here and there entirely escape, which are fewer in number the more severe the exposure. In the most exposed region, after twenty-four hours many of the nuclei are barely or not at all visible, while most of the others are in various stages of pycknosis and fragmentation. The cytoplasm often contains eosinphilic and basophilic granules similar to those seen in the lens epithelium. These are more abundant after twenty-four hours and are best seen in thin tangential sections. The eosinophilic granules are less readily seen in the cornea than in the lens epithelium, probably because they are to a greater or less degree masked by the eosin stained stroma. The effect on the corneal corpuscles is progressively less the deeper they lie, but an exposure of five minutes to the double lens system through the crown screen (295 $\mu\mu$) is sufficient completely to destroy all the corpuscles in the entire thickness of the cornea and also to destroy the endothelium. Polymorphonuclear leucocytes begin to invade the cornea in about twenty-four hours, reaching their maximum number in about

forty-eight hours. The purulent infiltration is greater the nearer the exposed area lies to the limbus, but is never sufficient to account for more than a small part of the haziness of the cornea. It is also greater the larger the area affected by the exposure.

The corneal endothelium in the most exposed region is entirely cast off within twenty-four to forty-eight hours. At the margins of the defect the nuclei show pycknosis and the cytoplasm often contains the characteristic basophilic and eosinophilic granules. These changes are also found after somewhat less intense exposures, in cells that remain adherent in exposed regions.

Repair of the Corneal Injury. Five days after exposure the epithelium is usually found reformed but thin. The visible corneal corpuscles are still further reduced in number, and of those visible many still contain eosinophilic and basophilic granules. Towards the periphery the nuclei are abnormally rich in chromatin and many of them enlarged. Some of them show direct division and budding. Occasionally a mitotic figure is seen here. The endothelium has not reformed. In places on Descemet's membrane there are eosinophilic and basophilic granules evidently left by necrotic endothelial cells.

After ten days the epithelium is still thin. The number of corneal corpuscles in the exposed area has slightly increased. The basophilic granules are apparently unchanged, but the eosinophilic granules in some cells stain less deeply and in others have apparently become confluent causing the whole cytoplasm to stain reddish. The nuclei are rich in chromatin, often polymorphous in shape, and sometimes show direct division and budding. Few if any mitotic figures are seen. The endothelium is completely reformed. After five weeks the cornea presents an almost normal appearance. The corneal corpuscles now slightly exceed the normal number. Many of the nuclei are abnormally large and a few cells contain double nuclei. The cause of the slight corneal opacity seen at this stage during life is not evident from the microscopic examination.

Histological Changes produced in the Cornea by Light rich in Abiotic Waves less than 295 $\mu\mu$ in Length.

With the bare magnetite arc, to destroy the epithelium of the cornea requires an exposure only one-eighteenth of that required when a crown screen (295 $\mu\mu$) is used. In the former case it is evident

therefore that the effect is due almost entirely to waves shorter than 295 $\mu\mu$. After an exposure of four minutes to the bare magnetite arc, at a distance of 20 cm. the epithelium, at the end of forty-eight hours, is entirely lost from the central two-thirds of the cornea. At the periphery the epithelium shows gradually increasing desquamation of its cells until it is reduced to a single layer for a variable distance and then abruptly ends. The cells even in the single layer are apparently not severely injured, and occasionally one is found in mitosis. They do not contain basophilic and eosinophilic granules, probably due to the fact that the shortest waves were absorbed by the superficial cells, while the remaining waves were not sufficiently intense at the periphery of the cornea to injure the deeper cells. The corneal corpuscles, lamellae, and endothelium are normal. There is, however, considerable purulent infiltration of the cornea. This is fully as great as in the case of exposures through a crown screen sufficient to injure the corpuscles.

Following an exposure of 20 minutes to the rays of the magnetite arc passing through a water cell and concentrated by the quartz double lens system, the following changes are seen: At the end of 10 hours the epithelium towards the periphery of the exposed area shows changes similar to those seen after exposure through a crown screen. As the central area is approached more marked changes occur; the nuclei are seen to become extremely pycknotic and the cytoplasm to stain intensely in eosin. Within the central area itself the superficial layers have become desquamated, leaving usually only the basal cells, which now consist of cylinders deeply stained in eosin from which the nuclei have entirely disappeared. Within the most exposed area at this stage the corneal corpuscles are still present in normal numbers. Their nuclei show marked pycknosis, but the cytoplasm contains no granules. Towards the periphery of the exposed area a few corpuscles containing granules are seen. The corneal stroma is swollen to a third more than its normal thickness, and stains less deeply in eosin. Unless the sections are very thick the stroma is apt to fall out of them. The individual lamellae are still recognizable but are greatly distorted, due no doubt to not holding their positions in the cutting of the sections. The endothelium is still adherent, but appears completely necrotic in the exposed area, the neuclei being pycknotic and the cytoplasm staining deeply in eosin.

After four days the epithelium is found to be reformed. Within the most exposed region the corneal corpuscles are completely invisible and the endothelium is absent. At the periphery of the exposed

area, many of the nuclei of the corpuscles are pycknotic or fragmented, and cells often contain eosinophilic and basophilic granules. Further away, the nuclei are enlarged and some of them show direct division. A few mitotic figures are also seen. The stroma in the exposed area is still more swollen, stains still less in eosin, and shows evidences of injury down to Descemet's membrane. The individual lamellae are no longer recognizable and the stroma appears as an almost homogeneous substance pervaded by indistinct wavy lines. There is a moderate leucocytic infiltration.

After twelve days (Pl. I, Fig. 1) the stroma is still more greatly altered. In the centre of the exposed area it has lost its normal structure and has undergone semi-liquefaction almost down to Descemet's membrane. This softened area contains a large amount of fibrin and a considerable number of pus cells and endothelial phagocytes. The leucocytes, however, are too few in number to cause an appearance in any way resembling an abscess. Around the area of softening groups of corneal corpuscles are actively proliferating, forming cells similar to fibroblasts. The epithelium is intact although altered in appearance. Numerous vessels are making their way into the cornea from the limbus. The endothelium has been almost completely reformed, but presents an abnormal appearance due chiefly to inequalities in the sizes and shapes of the cells.

After two months the cornea has returned to its normal thickness. The epithelium and endothelium are normal. The stroma in the affected region presents an abnormal appearance, but less so than might be expected. The corneal corpuscles are greatly increased in number and their nuclei are abnormally rich in chromatin. The new formed corneal lamellae are less regularly arranged than in the normal cornea and here and there occur areas of hyaline tissue that has not yet become definitely laminated. Blood vessels are still present but are small and few in number.

The Conjunctiva.

The clinical effects of exposure of the conjunctiva to abiotic rays have already been described. Histologically the following changes were noted in the bulbar conjunctiva 24 to 48 hours after exposure to abiotic radiations: Necrosis and desquamation of the epithelium. Infiltration of the epithelium with pus cells. Congestion, edema,

interstitial hemorrhages, and slight purulent infiltration of the subepithelial tissue. Basophilic and eosinophilic granules were not observed in the epithelium, possibly due to the fact that the cells were cast off when this degree of injury was reached. These changes were obtained after exposure to the magnetite arc with and without the single quartz lens. In the experiments with the double lens system the conjunctiva was not exposed.

The Iris.

Clinical. Twenty-four to forty-eight hours after an exposure sufficient to injure the lens epithelium, the pupil becomes contracted and the iris shows marked congestion and minute interstitial hemorrhages in the exposed region. The congestion quickly subsides, but the hemorrhages may remain visible for several weeks.

Histological. The iris is directly affected only after exposures sufficient to injure the lens epithelium. After exposure to the bare magnetite arc sufficient to produce marked conjunctivitis and keratitis, but insufficient to produce apparent injury the lens epithelium or corneal corpuscles, the anterior chamber may contain serum and fibrin, evidently the result of an indirect effect on the iris vessels. After exposures sufficient to injure the lens epithelium, there is seen, in addition to congestion and interstitial hemorrhages, an insignificant exudation of pus cells from the iris vessels. With these changes, few if any individual cells of the iris may show signs of injury. After an exposure of 20 minutes to the magnetite arc and lens system, the albinotic iris in one experiment (Exp. 68) shows marked cell changes similar to those of the lens capsule. The cells affected are the stroma cells, the endothelial cells of the vessels, and the posterior epithelium. From some of the blood vessels the endothelium is completely lost. Thrombosis, however, is not observed. The characteristic basophilic and eosinophilic granules are most noticeable in the cells of the posterior epithelium, no doubt due to the fact that these cells are most abundant. Similar changes are found after 6 days in a lightly pigmented iris (Exp. 56) but here the pigment hides any possible change in the pigment epithelium. In most of the experiments with the double lens system the pupil was widely dilated so that the iris was only slightly exposed to the light.

Posterior synechiae were not observed in any of our experiments.

Birch-Hirschfeld states that adhesion of the pigment epithelium to the lens occurred after fixation in some of his experiments, although the light intensities used were far less than those used by us. As already pointed out, the adhesions noted by Birch-Hirschfeld were undoubtedly artefacts due to the action of the fixing fluid alone, since they occur in the case of normal eyes. In view of the numerous control eyes examined by this observer, it is difficult to understand why he was not aware of this fact.

The Character of the Changes Produced in the Lens by Abiotic Radiations.

The light intensities and wave lengths necessary for the production of abiotic effects in the lens epithelium have already been given (page 651).

In none of our experiments was an opacity of the lens produced sufficient to be visible through the cornea. Even when the lens was examined in air after its removal from the eye it appeared perfectly clear. If, however, it was placed in normal salt solution it showed a delicate haziness in the pupillary area 48 hours after a severe exposure.

Histological. In all except one experiment upon the lens, the capsule was removed and examined as a flat preparation, so that it was impossible to make a satisfactory examination of the lens substance. To determine the effect of the abiotic radiations upon the latter, the lens in one experiment (Exp. 67) was fixed in formalin and horizontal sections made of it. The magnetite arc, water cell and system of quartz lenses were used without a screen, and the exposure was 20 minutes. This was the exposure that had been found to produce extreme changes in the capsular epithelium. The eye was enucleated at the end of 48 hours. On microscope examination the lens capsule proper is found unaltered, while the epithelium shows the marked changes described below. The lens substance is definitely affected but only for a microscopic depth, the distance beneath the capsule by actual measurement nowhere exceeding 20 μ. In this narrow zone it stains much more intensely in eosin than the rest of the lens substance and is highly vacuolated. Occasionally it contains an epithelial cell which has evidently been forced into it.

Lens Capsular Epithelium. This is the best possible tissue in which to study the cell changes produced by abiotic radiations because of

the simplicity of its structure and abundance of its cells, and because, by means of the flat preparations described (page 660) the whole of the exposed area may be examined at once. Moreover, the effects produced in it are not complicated by the presence of leucocytes, since these cannot penetrate it. Following are the histological changes produced in the epithelium by abiotic radiations:

If the capsule is fixed immediately after exposure, even if the latter has been prolonged, the cells appear absolutely normal. After 24 hours, changes are well marked, and reach their maximum in from 48 to 72 hours. After severe exposures, the cells may be so greatly affected that many of them no longer adhere to the capsule unless the latter is fixed within 24 hours. It is noteworthy that the cells in the exposed area are not all affected alike and one cell, or group of cells, may be markedly affected while the neighboring cells are only slightly affected. The chief changes noted consist in (a) swelling of the cells, (b) the appearance of granules in the cytoplasm, and (c) the formation of a peripheral wall of cells.

(a) After short exposures swelling of the cells may be almost the only change noted. It is plainly evident after an interval of 20 hours, but does not reach its maximum until after 48 hours. It is associated with increased transparency of the cytoplasm. All the cells do not swell to an equal extent, and as a result of the inequalities in compression the cells become misshapen in an irregular manner.

(b) The granules (Pl. 2, Figs. 5 and 6) in the cytoplasm first appear before the cells become much swollen. They are present within 10 hours but are more abundant after 48 hours. While evidently in the case of any individual cell a greater exposure is necessary to produce them than is required to produce swelling alone, a few cells containing them may always be found if the epithelium is affected at all. The longer the exposure the greater the number of cells containing them, and also the greater the number of granules in each cell, so that after prolonged exposures almost every cell may contain them. The granules are of two kinds. The more abundant are more or less strongly eosinophilic, usually round in shape and varied in size, the largest exceeding half the size of the nucleus. One cell may contain from one to over twenty granules. Each usually appears to be situated in a vacuole which it does not quite fill, but this may be due to shrinkage as a result of fixation. Close examination shows that they have a reticulated and subgranular structure. The other granules are intensely basophilic, and smaller than the eosinophilic granules,

the largest being about one-fifth the diameter of the nucleus and the smallest immeasurably fine. They also are usually round, but sometimes irregular in shape. Often they are contained within the eosinophilic granules. Owing to their strong basophilic character, the natural assumption would be that they represent chromatin extruded from the nuclei. Such an origin however, cannot actually be traced. On the contrary, the impression is given that the cytoplasm first breaks up into, or is transformed into the eosinophilic granules, and that the basophilic granules are formed primarily within the latter. After intense exposures, as will be pointed out, the nucleus may undergo disintegration, in which case some of the granules in the cytoplasm are undoubtedly nuclear fragments.

(c) The wall, first so named by Hess,[179] consists of a ring of deeply staining closely packed cells at the periphery of the exposed area, that is in the position corresponding to the pupillary margin at the time of the exposure (Pl. 3, Figs. 8 and 9). The cells are evidently in a state of compression and in marked cases may be heaped upon each other. The wall is visible after 19 hours but later becomes more evident. The cells within it show only to a slight extent the changes seen in the central area. Martin[238] assumed that the wall was due to "submaximal damage at the pupillary margin." This, however, is certainly not the case since submaximal exposures or any other exposures do not give rise to a similar condition of the cells within the pupillary area itself.* Hess explained the wall as a result of the compression of the marginal cells by the sheet of swollen cells in the pupillary area. This explanation seems undoubtedly correct. We have found a similar if not identical wall four days after the injection of staphylococci into the anterior chamber. The cells in the pupillary area were swollen but did not contain basophilic and eosinophilic granules. We have found such a wall also 24 hours after the injection of Lugol's solution into the anterior chamber, as described below (page 676). As will also be pointed out, a somewhat similar but yet different wall may be produced by the action of heat transmitted by the iris (page 696).

In spite of the marked changes in the cytoplasm of the exposed cells, the nuclei remain comparatively normal in appearance except

* Martin described in the capsule of one rabbit repeatedly exposed, a zone of proliferated cells which somewhat resembled the wall of Hess. The pupillary area, however, was otherwise free from abiotic changes. The condition was attributed to the effects of abiotic radiations, but in our opinion was almost certainly a congenital malformation such as we also have seen.

after the most intense exposures. Some nuclei show distortion, due possibly to the uneven compression of the swollen cells, and some stain less deeply than is normal, but following exposures through a crown screen fragmentation is seldom observed. Marked nuclear changes are seen after long exposure to the magnetite arc through the double lens system without a screen, but even then only relatively few nuclei are affected. Ten hours after such an exposure nuclei here and there show the following changes: The nucleus becomes transparent and its chromatin converted into coarse deeply staining granules attached to the nuclear membrane. The transition of a normal nucleus into this state is evidently very abrupt. The nucleus then becomes polymorphous in shape and undergoes fragmentation. Usually the fragments are each bordered by nuclear membrane and contain one or more coarse chromatin granules.

Mitotic figures are first seen after about 48 hours among the unexposed cells just outside the wall where they occur in large numbers. After 5 days they are greatly diminished in number here. After 3 days a few may also be found in the wall itself. Within the exposed area mitotic figures are not seen until about the fifth day when they occur in considerable numbers. At this time the cells are still swollen. The basophilic granules are little if any changed except possibly they are more often irregular in shape, but the eosinophilic granules have largely become confluent and are apparently undergoing solution. The mitotic figures are never seen in cells containing granules. Many of the nuclei are abnormally large and show early stages of direct division and budding.

At the end of ten or twelve days the cells have almost entirely lost their swollen appearance and the basophilic and eosinophilic granules have almost entirely disappeared. The most striking feature now consists in the inequalities in sizes and shapes of the nuclei. Most of the nuclei are abnormally large; occasionally one has three times the diameter of a normal nucleus. Some are abnormally small. Many of the nuclei evidently are undergoing direct division, as all the stages in this process can be seen, from a slight constriction of the nucleus to two nuclei connected by a delicate strand. In addition to this, a process of budding can similarly be traced, the nuclei becoming polymorphous in shape and constricting off buds varying in size from that of a normal nucleolus to half that of a normal nucleus. Some cells contain as many as twelve of these free buds. The buds have the reticulated structure, staining reaction and general appearance of the nucleus proper, and each most often contains a nucleolus. Cells

that contain two nuclei of nearly equal size always contain smaller buds in addition. At first glance the nuclear buds may be mistaken for persisting basophilic granules, but careful examination shows that they bear no relation to the latter either in appearance or origin. Few if any mitotic figures can now be seen in the exposed area or elsewhere.

At the end of 5 weeks or 2 months the capsule shows about the same appearances as after 10 days (Pl. 3, Fig. 7). There is perhaps still greater variation in the sizes of the nuclei, and a greater number of the excessively large ones. The cells with double nuclei and nuclear buds are still present. In case of the extremely severe exposures, a few cells are found still containing basophilic granules after two months.

In connection with the foregoing observations on the lens capsule several interesting questions arise. In the first place how is the abundant mitotic division of the unexposed cells in and around the wall to be explained? This proliferation is not due to minimal exposure to the rays for it does not occur in the pupillary area 48 hours after liminal or subliminal exposures. It is also not due to heat transmitted by the iris, because when a flint screen is substituted for a crown screen it does not occur after exposures more than four times as long. The only remaining possibility seems to be that it is due to toxic substances diffused from the injured cells of the exposed area.

If this is the case why are not mitotic figures seen at the same time in the exposed area? The answer to this is probably that the cells are here so greatly injured that they cannot respond at once to the irritation of the toxic substances, which, moreover, may at first be so concentrated as to inhibit rather than stimulate the nuclei. This brings up the question whether abiotic radiation is a direct stimulant or depressant to mitosis. It certainly is not a direct stimulant because, as just stated, after liminal or subliminal exposures mitosis does not occur. On the other hand it probably is a depressant because following intense exposures mitosis occurs in the exposed area only after relatively long intervals (four to five days) and then only in cells that have escaped apparent injury. This is in marked contrast to the action of heat, which, as will be shown, produces abundant mitosis in 48 hours and is evidently an active stimulant to cell proliferation.

Whether or not abiotic radiation is a direct depressant to mitosis

it is certain that repair of the injury to the lens epithelium takes place largely without the aid of this process. This is proved by the fact that mitosis does not occur in the severely injured cells, that is in the cells containing granules. Each of these cells, therefore, if it undergoes recovery as usually is the case, must do so without indirect division. It is evident that the eosinophilic and basophilic granules finally become dissolved out. The enlargement, direct division, and budding of many of the nuclei probably represent the response of the latter in the process of cell repair. Similar nuclear changes are sometimes seen in malignant tumors. The nuclear buds are still present at the end of two months and their ultimate fate is problematical.

Finally the question arises whether or not the cell changes described are characteristic only of the action of abiotic radiation. As will be pointed out later, experiments on the cornea prove that the basophilic and eosinophilic granules are not produced by heat, and thus their occurrence in cells constitutes a distinct difference between heat and abiotic effects. On the other hand, the following experiment proves that the same cell picture may be produced by chemical agents. A few drops of Lugol's solution containing 25% iodine were injected into the anterior chamber of a rabbit's eye. The injected fluid became mostly precipitated so that its action on the lens surface was not uniform. On examining the lens capsule 24 hours later there were found, in addition to more extreme changes, areas in which the cells showed identically the same changes, including the basophilic and eosinophilic granules as are produced by the action of abiotic radiation. It is therefore obvious that these changes are not characteristic of abiotic action alone, but may be produced by other forms of chemical action as well. It is interesting that in this experiment, as previously mentioned, a wall was formed similar to that produced by abiotic rays, evidently due to the pressure of the injured cells within the pupillary area on the peripheral cells which were protected from injury by the contact of the iris with the lens (Pl. 3, Fig. 10).

The changes just described occurring in the lens capsule after exposure to abiotic rays, are essentially the same as those described by Hess [179] who used much longer exposures but a light source of much less intensity than employed by us. Hess does not describe the granules in the cytoplasm, although they are shown well in his excellent illustrations. He also does not describe direct division and budding of the nuclei, although the latter process likewise seems to be

shown in one of his illustrations. Apparently he attributed the repair of the injury chiefly to mitosis and not to recovery of the injured individual cells. He states, however, that he has no evidence that ultra violet light is a direct stimulant to mitosis. Widmark,[418] strangely enough, found mitotic figures only in the exposed area and regarded ultra violet light as a direct stimulant to cell proliferation.

Birch-Hirschfeld[38] states that by means of a 20 diopter glass lens he focussed the light of a 5 ampere arc light through a euphos glass screen upon the eye of a rabbit for five minutes for three successive days and on the day after the last exposure obtained the changes described by Hess. The euphos screen obstructed all rays less than 400 $\mu\mu$ in length. It is not stated that a water cell was used, and the diameter of the lens was not mentioned. In spite of such a remarkable result it is not stated that the experiment was repeated. We have been unable to obtain such a result through a light flint screen transparent for waves down to 315 $\mu\mu$ with the magnetite arc and still greater concentration of energy. Moreover in an experiment in which we focussed sunlight upon the lens by means of a large mirror no changes in the lens capsule resulted within the pupillary area, although there was complete necrosis of the iris due to heat. The lens capsule was affected only beneath the pupillary margin where it had been in contact with the heated iris and even here the changes were not such as are produced by abiotic action. We are therefore compelled to believe that Birch-Hirschfeld was in error. Possibly he mistook a heat effect similar to that just noted for the changes described by Hess. He had never previously obtained the latter changes in any of his experiments and hence from personal observation was no doubt unfamiliar with their appearance.

POSSIBLE ABIOTIC EFFECTS OF RADIANT ENERGY ON THE RETINA.

It might be supposed that if a source of light is not sufficiently rich in abiotic rays to damage the cornea, the retina could not be injured by these rays. This, however, is not necessarily true because if the source of light is so small in size that the area of its retinal image is less than that of the pupil, the intensity per unit area as concerns transmissible rays will be greater on the retina than on the cornea.

In fact under certain conditions, and with a moderately dilated pupil the intensity of the light reaching the retina will be enormously greater than the same light as it passes through the cornea. For this reason it will be seen that if the transmissible rays were capable of injuring tissue cells, the macula of the eye might be seriously damaged in spite of the fact that the cornea and lens remained unaffected. This, of course, actually happens in eclipse blindness in which, however, as will be pointed out, the effect is due entirely to heat generated in the pigment epithelium.

There are two conceivable ways, exclusive of heat effects, in which the retina could be injured by light. If the light were sufficiently intense it might overstimulate the physiological mechanism upon which the perception of light is dependent and thus lead to more or less permanent impairment of this mechanism. It is obvious that such an effect could not readily be produced by light of wave lengths less than 400 $\mu\mu$ since the latter has relatively little power to stimulate this mechanism even in aphakic eyes. The other possibility is that intense light might injure the cells of the retina by abiotic action in the same way that light rays of short wave length injure tissue cells in general. In connection with this possibility two facts previously established by us must be taken into consideration, namely that within wide limits discontinuous exposures to abiotic rays have the same total effect as a continuous exposure of the same total length, and that there is a limit below which such summation does not occur. Thus it would *a priori* seem possible that if an individual fixed a bright source of light many times daily, serious damage to the macula might result.

The problem in regard to the retina that chiefly concerns us in the present investigation may be briefly stated thus: exclusive of a heat effect, can the retina of the human eye be injured by light of any or all wave lengths that can possibly reach it through the cornea and lens? In attempting to answer this question it is important first to inquire whether or not the waves that are able to pass through the dioptric media are injurious to tissue cells in general. If they are so injurious the question is obviously to be answered in the affirmative. If they are not, the question is in all probability to be answered in the negative, but not perhaps with absolute certainty, since it is conceivable that the retinal cells are more susceptible to injury by light than are other tissue cells.

It has been shown by Hallauer [152] and others that the adult human lens always absorbs all waves less than 376 $\mu\mu$ in length, and usually

all those less than 400 $\mu\mu$ in length. Now we have already shown that the corneal epithelium and lens capsule are not affected in the slightest degree when exposed one and one-half hours to rays as short as 310 $\mu\mu$ even when the intensity is considerably greater than that to which the retina is ever subjected in the case of any of the known artificial light sources. This exposure is at least forty-five times greater than that required to affect the corneal epithelium by waves of 295 $\mu\mu$ and less. For the retina therefore to be affected by the abiotic action of light transmitted by the lens, it would have to be many times more sensitive to such action than the corneal epithelium. There is no reason to believe however, that this is the case, but on the contrary, since the abiotic effect depends upon the amount of absorption of the waves, there is strong reason for believing that the corneal epithelium and retina are about equally sensitive to abiotic action. Assuming this to be so, these experiments show conclusively that the human eye could be fixed steadily and at close range upon the magnetite arc certainly for over two hours and probably for many hours without suffering damage to the retina from abiotic action. Since as already pointed out the intensity of the image of a source of light of such small size as that in question decreases as the square of the distance, the danger of injury to the retina at ordinary distances would be absolutely negligible.

In the case of the lenses of some children, Hallauer found a very weak transmission band at 315 to 330 $\mu\mu$. This, however, does not invalidate the application of the above argument to the case of children, since we have shown that such waves are without abiotic effect.

While it seems to us that the foregoing facts prove conclusively enough that the lens affords complete protection to the retina from the abiotic action of light, in view of the fact that Birch-Hirschfeld claims to have produced pathological changes in the retinae of normal rabbit's eyes by exposure to ultra violet light (cf. page 687), we have undertaken to investigate this question by direct experiment upon the retina itself.

Such an investigation presents several difficulties. In the first place it is impossible to reproduce with animals exactly the conditions that obtain when the human eye is fixed upon a small intense source of light. This is so because it is impossible to insure in the case of an animal that the small image of the light source will always fall upon the same spot in the retina during the exposure. Moreover, even if this were possible it would be difficult if not impossible to find with certainty such a small area on microscopic examination unless

the lesion produced was well marked. It is therefore necessary to illuminate a large area of the retina. This we have done by means of a suitable system of quartz lenses used in connection with the magnetite arc as described on page 648. Intense illumination of such a large area, however, for a long period of time entails a danger of overheating the fundus of the eye. This we have successfully obviated by interposing a quartz cell 5 cm. thick filled with distilled water to absorb most of the infra red rays. If this had not sufficed, heat effects could probably still have been prevented by interrupting the exposures at intervals to allow for cooling to take place, a procedure that no doubt would be necessary for light intensities only slightly greater than that used by us. In fact in one of these experiments in which the water cell leaked, a heat effect on the pigment epithelium was actually noted (Exp. 88).

As will be seen the system of quartz lenses employed concentrated the light more intensely upon the cornea and lens than upon the retina. Advantage was therefore taken of this fact to determine at the same time the effect of exposures through various screens upon these structures, the results of which have already been given. None of the screens obstructed any waves longer than 305 $\mu\mu$ to 315 $\mu\mu$, that is, any waves that otherwise could have reached the retina through the lens. The screens also prevented excessive keratitis, which we desired to avoid since it would have prevented us from later making satisfactory tests of the lid reflex and pupillary reaction to light. If these reflexes had been abolished this fact alone would have furnished sufficient proof of the deleterious action of the radiations on the retina. As a matter of fact, except immediately after exposure a lid reflex was always obtainable.

The details of these experiments are given on pages 655–658. (Experiments 65 to 90). It will be observed that the exposure was as long as one and one-half hours in each of four experiments and one hour in each of six experiments. In all except one experiment the retinae were prepared for microscopic examination in the manner already described (page 661). In Experiment 78 the eye was immediately opened and the retina bisected vertically through the optic disc, one half being fixed in a saturated solution of mercuric chloride and embedded in paraffin. The sections, 2 μ in thickness, were stained in thionin as in the case of the other experiments. The other half of the retina was used for vital methylene blue staining, the results of which will be mentioned later in commenting on Birch-Hirschfeld's observations (page 687). In none of the experiments could any

apparent changes be found in the exposed retinae that could not be found in unexposed retinae prepared by the same method. Certainly if there were any differences in regard to the Nissl bodies of the ganglion cells they were too slight to be of any pathological significance.

These experiments thus show that, so far as can be determined by histological examination, the retina of the normal eye, exclusive of heat effects, is fully protected from abiotic action by the lens. Since, however, the objection may be brought forward that the retina may be injured so far as its function is concerned without showing any histological evidence of the fact, we have endeavored to exclude this possibility also. For this purpose we employed the monkey instead of the rabbit because this animal possesses a macula similar to that of man. With the lens system described it is easily possible to illuminate intensely a sufficient area of the retina to insure that the macula is always included. If under these circumstances the light has an injurious action on the retina it will be rendered evident, since the macula is injured, by marked impairment in sight and particularly by a loss or impairment of the pupillary reaction. To avoid injury to the cornea by abiotic rays and injury to the retina by heat we made use of a $1\frac{1}{2}\%$ solution of copper chloride in a quartz cell 5 cm. thick. The spectrum of this solution (Pl. 5, Fig. 4) shows that it absorbs all waves shorter than 320 $\mu\mu$ as well as all the so-called heat waves. It thus does not obstruct any short waves that could otherwise reach the retina through the lens.

In these experiments two monkeys were employed in each of which the left eye was blind. One was an old female monkey, whose left eye had been rendered blind by an experimental Krönlein operation involving injury to the optic nerve, one year previous to the first of the present experiments. The other was a young full grown male monkey, whose left eye had been rendered blind by injection of alcohol into the orbit nine months previous to the first of the experiments. Ophthalmoscopic examination showed complete optic atrophy in the left eye of each. Neither monkey could find the way about when the right eye was excluded from vision. Direct pupillary reaction to light was absent, but the consensual reaction was well marked. This made it possible to determine the presence of a pupillary reaction while the right eye was under the influence of the mydriatic, while the fact that the left eye was blind made it easy to detect any impairment of vision of the right eye. In each animal the visual acuity of the right eye was high, as shown by the ease with which it was able to catch flies and lice. The absence of binocular vision did

not seem to hamper either animal in its judgment of distance except for a short time after the left eye had been made blind.

In all four experiments the magnetite arc was used and the same arrangement of lenses employed as in the previous experiments with rabbits, the quartz cell, as stated, being filled with a $1\frac{1}{2}\%$ solution of copper chloride. The light was focussed on the centre of the cornea. The animal was placed in a box which allowed only the head to protrude, and the eyelids kept open by means of a small speculum. The head was forcibly held in position by the hand of the observer. For the first five minutes the animal was difficult to control, but after this no great difficulty was experienced in keeping the eye in place. No local or general anaesthetic was employed. Normal salt solution was dropped on the cornea from time to time. The pupil of the right eye was previously dilated by homatropine except in the third experiment in which atropine was used.

To give some idea of the light intensity and duration of the exposures in these experiments, it may be well to state that one of us exposed his eye with undilated pupil to these conditions for fifteen seconds, and obtained an absolute scotoma which gradually disappeared within five minutes. Erythropsia persisted about three minutes and was followed by xanthopsia which lasted the remainder of the five minutes.

Experiments.

Experiment 94. March 7, 1913. Young monkey, *Macacus Rhesus*. Right eye exposed $1\frac{1}{2}$ hours. Immediately after exposure there is a lid reflex to a new $2\frac{1}{2}$ volt tungsten flash light, and within five minutes the consensual pupillary reaction is apparently normal. Within ten minutes the animal is able to see an apple five feet away, which he approaches and takes from the hand. March 8. Cornea clear. Consensual pupillary action normal. Slight direct pupillary reaction in right eye in spite of mydriasis. Owing evidently to the cycloplegia, the animal cannot catch flies readily. After several days, the mydriasis having disappeared, the animal is able to catch flies with his usual dexterity.

Experiment 94a. March 28, 1913. Old female monkey. (Java.) Right eye exposed $1\frac{1}{2}$ hours. Immediately after exposure the lid reflex to the flash light is absent, but is present in five minutes. Consensual pupillary reaction not determinable owing to some of the

mydriatic having accidently gotten into the left eye. Animal has great difficulty in getting around, vision evidently being much impaired. After one hour vision is much improved; the animal follows the observer with the eye. March 29, 1913. Cornea clear. Well marked lid reflex to flash light. Consensual pupillary reaction also well marked. Visual acuity of animal apparently normal except that animal has difficulty in catching flies owing to effect of cycloplegia. After several days, the mydriasis having disappeared, the animal is able to catch flies with her usual dexterity.

Experiment 94b. December 4, 1913. Old female monkey. Right eye exposed 1½ hours. One minute after end of exposure there is a barely perceptible consensual pupillary reaction. After six minutes the reaction is well marked. Animal now released. Cannot see approach of observer's hand. Is compelled to feel her way to her perch in the cage. After one hour she is still apparently blind; cannot see a carrot held near her, although she takes it when placed against her mouth. After seven hours, vision is still impaired. December 5, (18 hours). Cornea clear. Vision apparently normal — sees carrot, avoids hand movements etc., even in poorly illuminated cage. Consensual pupillary reaction normal. After the mydriasis has disappeared the animal catches flies as usual.

Experiment 94c. February 5, 1914. Young monkey. Right eye exposed 1½ hours. Three minutes after beginning of exposure the consensual pupillary reaction, tested with flash light, is absent. Immediately after end of exposure the lid reflex to flash light is present, but the consensual pupillary reaction is absent. At the end of three minutes the latter is distinctly visible, and in six minutes is well marked. Animal now released, finds his way at once to perch, avoids hand of observer — evidently sees well. One hour after exposure the eye lids of right eye are sewed together. When released the animal cannot find his way about and is easily caught, thus showing that if the sight of the right eye had been affected the fact would have been easily determined. February 6. There is a small abrasion of the cornea probably due to the animal having frequently rubbed his eye as a result of a slight irritation produced by the sutures. Cornea clear. Consensual pupillary reaction intact. Animal sees well. February 7. The abrasion of the cornea is healed. Consensual pupillary reaction intact. Animal shows no evidences of poor vision. After the mydriasis has disappeared the direct pupillary reaction to light is normal and the animal seems to have normal vision.

The results of these experiments show that even with exposures of

extreme intensity and length, but insufficient to produce heat effects, it is impossible to injure the retina by light containing any or all rays capable of reaching it through the lens. They exclude both the possibility of injuring the retina by over stimulating its perceptive mechanism, and also of injuring it by the abiotic action of light. Most surprising was the rapidity with which the retina regained its function. Thus in all four experiments within six minutes the consensual pupillary reaction was fully reestablished. There was in both sets of experiments, however, a marked difference between the young and the old monkey in regard to the time required for the restoration of useful vision. In the case of the young monkey sufficient vision to enable him to see his way about, avoid hand movements, etc., was present in ten minutes after the exposure ended. The old monkey on the other hand was practically blind for an hour or more. In fact her visual acuity did not seem to be fully restored until the morning following the exposure. Both animals were able to catch flies with their usual expertness after the mydriasis had disappeared.*

The results obtained in these experiments would also seem to be of some significance in regard to the question of light adaptation. They suggest that after a certain state of retinal fatigue is reached no further effect is produced, however long the exposure. In fact it would seem, in young individuals at least, that after this stage is reached the recuperative processes begin while the retina is still exposed. This aspect of the question, however, does not concern us here and further experiments would be necessary to elucidate it fully.

In addition to the experiments on the eyes of monkeys we have availed ourselves of an exceptional opportunity to make a similar experiment upon a human eye. The subject was a female patient aged 50 years affected with carcinoma of the eyelid and orbit, the growth being so extensive as to necessitate removal of the eye. The left eye itself was apparently normal, the media being clear and the fundus normal. The visual acuity was reduced to $\frac{20}{30}-$ (unimproved by lenses) for some reason not definitely determined, but probably due to some irregularity in refraction resulting from the pressure of the upper lid. The lower lid was almost completely destroyed, while the upper lid was somewhat drawn down by cicatricial tissue at the outer canthus. It was therefore necessary for the observer to hold up the eyelid by finger pressure during the experiment. The right

* Both of these monkeys were later killed, one after seven months, the other after fourteen months, and on microscopic examination the eyes that had been exposed were found normal.

eye was normal and had normal visual acuty. Before the experiment the pupil of the left eye was dilated with atropine, but the visual acuity remained the same. The total exposure was less than in the case of the monkeys, owing to the patient becoming somewhat fatigued, and for the same reason also the exposure was not continuous, but otherwise the conditions of the experiment were the same.

The total exposure was 55 minutes, and the interval between the separate exposures was about $1\frac{1}{2}$ minutes. The first three exposures were 3, 9, 12 minutes respectively, the remainder were 5 minutes each. At the beginning of each exposure the patient stated that the "light was like the sun." At the end of the sixth exposure there was erythropsia and the visual acuity was reduced to counting of fingers at one foot. Within $2\frac{1}{2}$ minutes after the last exposure the consensual pupillary reaction was well marked, and the patient could with difficulty count fingers at six feet. Three minutes after the last exposure there was only slight erythropsia. Xanthopsia was not noted at any time but may have been unrecognized by the patient. After 10 minutes the visual acuity was $\frac{20}{200}$. There was an appearance of a mist before the eye, but no erythropsia. After $1\frac{1}{2}$ hours the visual acuity was $\frac{20}{100}$, and a slight mist still persisted. After 3 hours the visual acuity was $\frac{20}{40}+$, and a white surface seemed almost but not quite as white as with the right eye. After 22 hours (in the morning), the visual acuity was $\frac{20}{30}-$ as before the experiment. There was no erythropsia, and central color vision was perfect for red, blue and green. 24 hours after the exposure the eye was enucleated. On microscopic examination the cornea, iris, lens epithelium (flat preparation), and retina were found to be normal.

The result of this experiment confirms those obtained with the monkeys. It is obvious that the retina could not have been injured by abiotic action of light, since the visual acuity was fully restored within 3 hours and remained so the following morning. The rapidity with which the erythropsia disappeared was unexpected, and indicates that duration of exposure is equally as important as its intensity in the production of persistent erythropsia.

POSSIBLE EFFECTS OF ABIOTIC RADIATIONS ON THE RETINAE OF APHAKIC EYES.

Since it has been shown experimentally that abiotic waves may pass through the cornea and injure the lens epithelium, it would seem that exposure of an aphakic eye to a light source rich in such waves might seriously damage the retina. Assuming as is probable, that the retina has the same susceptibility to abiotic action of light as the lens epithelium, the minimal exposure to the bare magnetite arc necessary to injure the retina of an aphakic human eye may be closely approximated from the data of our experiments. The working diameter of the single quartz lens was 4.2 cm., and the working focal distance 14 cm., making the working aperture 1/3.3 This corresponds to the aperture of a human aphakic eye with a pupil 4.5 mm. wide. Now we found that the lens epithelium of a normal rabbit's eye was unaffected by an exposure of 6 minutes to the single lens system through a crown screen (295 $\mu\mu$), but moderately affected by an exposure of 12 minutes. The liminal exposure may therefore be taken as 8 minutes. The total loss by reflection etc. from the surfaces of the lenses, screen, and water cell, amounts to about 50%. Deducting this percentage, the minimum exposure to the magnetite arc necessary to affect the retina of a human aphakic eye would therefore be about 4 minutes, providing that the eye was close enough for the formation of a distinct image, and ignoring the blurring due to the lessened refraction of an aphakic eye. The absorption of the cornea is allowed for in this calculation, since in the experiments the light passed through the cornea, but the general absorption of the vitreous humor is not. Assuming this to be about the same as that of the cornea (although it probably is greater) the calculated exposure would be increased to about 6 minutes. Since beyond $1\frac{1}{2}$ meters, owing to the small size of the source, the intensity of the light on the retina would diminish as the square of the distance, it is safe to say that under the most favorable conditions, it would require fixation of the bare magnetite arc at a distance of 3 meters for almost $\frac{1}{2}$ hour to injure the retina of an aphakic eye. According to our experiments on the effects of repeated exposures (page 641) a daily total exposure of $\frac{1}{3}$ the liminal, which in the present case would be 8 minutes at a distance of 3 meters from the magnetite arc, would produce pathological effects in the retina of the

aphakic eye in 6 days, while a total daily exposure of $\frac{1}{6}$ the liminal, in this case 4 minutes, would produce no effects even if indefinitely continued. These estimates do not allow for the pupillary contraction, which would result from the fixation of such a bright source and which would in most cases increase the necessary exposures three or four times, or for imperfect fixation. They also do not allow for the thick cataract glasses which in most cases would be worn and which would increase the necessary exposures many times, since a 10 dioptre lens would be almost impenetrable to abiotic radiations. For the quartz mercury vapor lamp still longer exposures would be required owing to the size and shape of the light source giving less concentration in the image. It is, therefore, now apparent why there is no known case of a human eye from which the lens has been removed in which the retina has been injured by exposure to artificial light, and why such injury is in the highest degree improbable.

In endeavoring to demonstrate by direct experiment the possibility of injuring the retina of the aphakic eye by abiotic radiation we have found it difficult to obtain a satisfactory eye for the purpose. While it was easily possible to remove the lens from the rabbit's eye, the pupil became more or less completely obstructed in almost all cases. In one animal, however, we finally obtained by means of repeated discissions, a clear pupillary opening sufficient to admit the cone of light from the quartz double lens system. According to our calculations an exposure of 35 minutes with the light focussed upon the pupillary area should have been sufficient to produce abiotic effects in the retina. No allowance, however, was made for absorption by the vitreous humor. As a matter of fact no abiotic effects could be demonstrated in the retina although marked heat effects were obtained in the pigment epithelium (Exp. 89). This experiment thus goes to show that the danger to the retina from exposing the aphakic eye to abiotic radiations is even less than is indicated by the above calculations.

Birch-Hirschfeld's Observations.

Since the results of our experiments especially in regard to the retina are so greatly at variance with those of Birch-Hirschfeld [37] it may be well to review his experiments in some detail. This is all the more necessary because his results and conclusions have not hitherto either been confirmed or refuted. His experiments consist of two series. In the first series he separated out the ultra violet

rays from a 15 ampere carbon arc lamp by means of a quartz lens and quartz prism, and concentrated them upon the anterior focal point of the rabbit's eye by means of a second quartz lens. The diameter and focal length of the latter he did not mention. He exposed both normal eyes and eyes from which he had extracted the lenses. The latter were seven in number. The length of the exposures were from one-fourth hour to 6 hours. Following the exposure there was only slight hyperemia of the conjunctiva which disappeared in 24 hours. The cornea and lens were unaffected even after the 6 hours' exposure. The retina on microscopic examination showed the following changes: chromatolysis and formation of vacuoles in the cytoplasm of the ganglion cells. Loss of chromatin in both nuclear layers, the nuclei of the outer layer becoming homogeneous and their cross striations almost completely obscured. These changes were found just the same immediately after exposure as in the course of the next 12 to 24 hours. After a few days they disappeared and the ganglion cells showed an increased amount of chromatin. In the animal which was exposed for 6 hours, however, vacuoles were found in the ganglion cells at the end of 6 days. In the case of normal rabbits' eyes exposed to the same conditions, retinal changes were found only when the eye was removed immediately after exposure and were said to be simply those of light adaptation.

In the second series of experiments he exposed the rabbits' eyes to a 3 to 4.5 ampere Finsen light. No statement is made as to whether or not a quartz lens or water cell were used, so it is to be presumed they were not. Also no statement is made as to the distance between the eye and the light. Ten eyes altogether were exposed, two being aphakic. The time of exposure was from five to ten minutes.

In all cases there was marked conjunctivitis, keratitis, and iritis (?), but no changes were ever found in the lens capsular epithelium. In the retina the following changes were found in both the normal eye and the aphakic eye, but were more pronounced in the latter: chromatolysis and formation of vacuoles in the cytoplasm of the ganglion cells with changes in the nuclei of the latter. Swelling and beginning collapse of the nuclei of the inner nuclear layer. Loss of chromatism in the outer layer. The vacuolization of the ganglion cells in some cases persisted several weeks.* When a thick glass plate

* In a footnote Birch-Hirschfeld stated that in one aphakic eye after an especially severe exposure to the iron arc he obtained well marked myelin degeneration of the optic nerve. He also stated that he would later give the details of this experiment, but we are unable to find that he has done so up to the present time.

was placed before the eye these changes did not occur. The exact length of time after exposure when the eyes were examined is not stated. It also is not stated whether or not the changes could be found if the eyes were removed immediately after the exposures.

It is impossible for us to accept the findings in these experiments for the following reasons. In the first place the retinal changes described were widespread. To obtain a widespread illumination of the retina, however, necessitates the use of a quartz lens of extreme aperture. Birch-Hirschfeld does not state that he used such a lens. The widespread illumination of the retina also necessitates a greater intensity of illumination of the cornea and lens than of the retina. This together with the fact that the lens capsule receives in addition rays of much shorter wave length than can reach the retina makes it inconceivable that the retina could be injured under these conditions without the lens capsule also being affected. Yet Birch-Hirschfeld states that in neither series of experiments was the intensity and duration of exposure sufficient to injure the lens. In fact the abiotic intensity was so slight that the corneal epithelium was destroyed only in one experiment in which the cornea apparently became infected.

On the other hand if we assume that Birch-Hirschfeld used no lens or a lens of ordinary aperture, the retinal lesions, if any, would have been circumscribed and would not often have been found by his method of examining the eyes. As we shall show, with sufficient light intensity small retinal lesions can be produced under these conditions, but they are due to heat and are entirely different from those described by Birch-Hirschfeld. The radiant energy used by Birch-Hirschfeld, however, was undoubtedly insufficient to produce such an effect.

In his first series of experiments it is stated that the changes occurred immediately after the exposures. This is inconsistent with an abiotic action of light, since with this there is always a latent period. Thus we found that the epithelial cells of the lens capsule showed absolutely no change if examined immediately after severe exposure to abiotic rays.

Birch-Hirschfeld holds that the ganglion cell changes he describes represent a further stage of light adaptation. Yet he maintains that they are due to the direct action of the light on the ganglion cells themselves. He states that there is no reason to believe that certain cells are more susceptible to ultra violet light than others, yet he found changes in the retinal ganglion cells and none in the capsular epithelium in spite of the fact, just pointed out, that the latter must have

received light not only of greater intensity but also of shorter wave length than did the retina.

We cannot then accept Birch-Hirschfeld's findings because we were unable to obtain retinal changes, although we used light intensities and exposures sufficient to injure the epithelium of the lens capsule, the stroma cells and endothelium of the cornea (which he did not). Judging by the relatively slight histological changes found in the cornea by Birch-Hirschfeld, the intensity on the cornea of the light used by us must have been over fifty times as great as that used by him, while some of our exposures were nine times as long as his maximum exposure (10 minutes) to the iron arc. In the case of the aphakic eye, we obtained no ganglion cell changes in 48 hours although the light intensity was so great that the pigment epithelium showed heat changes in spite of an interposed water cell. In Birch-Hirschfeld's experiments the pigment epithelium was uninjured although a water cell was not used. Finally, we cannot accept Birch-Hirschfeld's findings because our experiments on monkeys and on a human patient prove conclusively that the function of the ganglion cells is not injured by light of the same wave lengths and vastly greater intensity than that reaching the retina in Birch-Hirschfeld's experiments.

In connection with Birch-Hirschfeld's findings the following observations relating to the ganglion cells of normal rabbit's eyes may be of significance. In the first place, within the same retina there is great variation in the amount of chromatin substance in the individual ganglion cells; two cells side by side may show a great difference in this respect.* The sharpness with which the Nissl bodies stain in thionin varies considerably with slight variations in the staining procedure, particularly as regards the length of time the sections have been immersed in the thionin solution and the degree of differentiation in alcohol. The same statement applies also to the intensity with which the nuclear layers stain. While the ganglion cells of a normal retina probably never contain actual vacuoles, the arrangement of the chromatin particles is not infrequently such that they enclose spaces which bear considerable resemblance to vacuoles. Occasionally a ganglion cell may contain an apparently degenerated nucleus, and occasionally also a more or less disintegrated ganglion cell is seen. Possibly the injury to the latter is produced by the microtome knife.

* Nissl bodies cannot be seen in fresh ganglion cells so that it is possible that they are formed after death. The term chromatolysis may therefore be misleading inasmuch as it means solution of substances during life which may have never actually existed.

These observations are in accord with those of Bach made twenty years ago. Investigating the possible effect of fatigue upon the ganglion cells of the retina, Bach [14] made a careful comparison of retinae of rabbits exposed and unexposed to light. In some cases eyes were exposed to a Welsbach light for 20 hours. Alcohol or sublimate fixation was used and the sections stained by the original Nissl method or in thionin. Contrary to the previous observation of Mann [235] and the later observations of Birch-Hirschfeld,[39] he was unable to find that the ganglion cells of the exposed eye differed in any way from those of the unexposed. He says: "Ich geh zu, dass ich längere zeit im Zweifel war und bald diese bald jene Veränderungen gefunden zu haben glaubte, jedoch alles anscheinend Gefundene liess mich die controle wieder als Irrthum erkennen. Es ist eben zu bedenken, dass trotz gleicher Schnittdicke, trotz des genau gleichen Verfahrens beim Färben etc. immerhim sich tinctorielle Unterschiede ergeben können..... Ich muss bemerken, dass auch in normalen Netzhäuten an den Ganglienzellen sich Unterschiede besonders hinsichtlich der Menge und Anordnung, der Form der färbaren Plasmaschollen ergeben, das auch normalen Weise Vacuolen in dem Zellleib gefunden werden, das die Kerne sich verschieden verhalten können — kurz ich konnte an den beleuchteten Netzhäuten Nichts warhnehmen oder vermissen was ich nicht an normalen, an verdunkelten Netzhäuten auch wahrgenommen oder vermisst hätte."

Birch-Hirschfeld [37] states that also by means of the vital methylene blue staining method he found chromatolysis, vacuolization, and other changes in the retinal ganglion cells of eyes that had been exposed to ultra violet light, and that such changes were absent in normal eyes. I find, however, by this method in the normal rabbit's retina, appearances that correspond exactly to those described and depicted by Birch-Hirschfeld, including particularly the "vacuoles" in the ganglion cells which are abundantly present. The "vacuoles" at first glance appear to be really such, but a careful study of them strongly suggests that they are here due to the cell reticulum staining more promptly and deeply than the cytoplasm proper, and thus producing an appearance of rounded spaces. I have also examined the retina by the vital methylene blue method 48 hours after the exposure of one hour to the magnetite arc and lens system (Exp. 78). The results obtained were identical with those obtained in the case of an unexposed normal retina.

In addition to the experimental investigation just discussed, Birch-Hirschfeld [35] has reported clinical observations in five cases of pho-

tophthalmia following exposure to the mercury vapor lamp that he claims demonstrate the pathological action of ultra violet light upon the retina. In these cases he found, for colors only, para- or pericentral scotoma, central relative scotoma, and constriction of the peripheral field. Later [24] he reports that after an exposure of less than $\frac{1}{2}$ hour to the Schott uviol lamp he himself was affected with mild photophthalmia followed by color field changes. He found a relative color scotoma in each eye beginning 15° from the fixation point that persisted 6 days. After this had completely disappeared he exposed his left eye to the same light through a colorless glass obstructing all waves less than 330 $\mu\mu$ in length and obtained no changes of any kind. He regarded this as proof of his contention that the field changes previously obtained were due chiefly to waves between 300 $\mu\mu$ and 330 $\mu\mu$. As a matter of fact, however, Hallauer [123] has shown that the adult human lens absorbs all waves less than 376 $\mu\mu$ and most of those less than 400 $\mu\mu$ so that this experiment of Birch-Hirschfeld proves, if it proves anything, that the field changes obtained in his clinical cases and in his own case were chiefly subjective or at least did not represent pathological conditions. Moreover, as pointed out elsewhere (page 721) Birch-Hirschfeld [41] himself has recently taken exception to the similar field changes reported by Jess [200] in cases of eclipse blindness on the ground that they might well have been obtained in normal eyes.

Thermic Effects of Radiant Energy on the Eye.

The Cornea. In passing through the cornea, light of any wave length is absorbed to some extent. Waves less than 295 $\mu\mu$ are completely absorbed while those over 315 $\mu\mu$ in length (judging by the results of our experiments) are very slightly absorbed. The absorption of the latter is no doubt due in part at least to the lamellae of the cornea and the corneal corpuscles, which cause internal reflections and refractions, especially of the relatively short waves. With ordinary light intensities the amount of energy absorbed is so slight that no heat effects are produced, but with extreme intensities it is obvious that the latter could be produced even in the case of visible rays. In five of our experiments definite heat effects were observed in the cornea. That the effects were due solely to accumulated heat and not in any degree to abiotic action, is proved by the character of the changes

produced, and by the fact that the epithelial cells of the cornea and lens were unaffected. The screens were such that the lens received waves of the same wave lengths as did the cornea. The corneal epithelium was unaffected probably owing to its being cooled by contact with the air. In no instance did the heat reach sufficient intensity as to cause pain.

The most marked heat effect on the cornea was obtained in Exp. 88 in which the rays from the magnetite arc after passing through a flint screen and water cell were concentrated for one hour sharply upon the cornea by means of the quartz double lens system. Toward the end of the experiment it was discovered that the water cell had leaked, so that for an unknown length of time the eye had been exposed to infra red rays in addition to the shorter waves. This undoubtedly accounts for the fact that in no other experiment was such a marked heat effect produced, and that no effect was produced in Exp. 85 in which the same conditions obtained except that the water cell did not leak and the exposure was longer. 24 hours after the exposure the affected area was hazy and swollen but the eye was free from inflammatory reaction. On microscopic examination 48 hours after the exposure the epithelium was everywhere normal. The stroma was swollen to over twice its normal thickness and stained faintly in eosin. Within the central portion of the exposed area not a single corneal corpuscle could be seen. At the periphery the transition into normal cornea was abrupt as regards the corpuscles but relatively gradual as regards the stroma. In the transition region the corpuscle towards the normal side were in active proliferation, many of them showing mitosis, while from here inward they suddenly became invisible. The endothelium in the exposed region was for the most part completely absent, but in some places a few faintly stained cells still adhered to Descemet's membrane. The cornea was everywhere practically free from leucocytic infiltration. The iris showed a few minute hemorrhages around the pupil undoubtedly due to heat, since, as stated, the lens capsule was unaffected. (Pl. 1, Fig. 3.)

In the second experiment (Exp. 92) sunlight was focussed 45 minutes upon the cornea by means of a large quartz lens after passing through a blue uviol screen and a .001% aqueous solution of auramine O. Here the heat effect was similar but less marked than that just described. The effect on the corneal corpuscles was about as great, and the appearance of the stroma about the same except that it was much less swollen. The corneal epithelium, the iris, and the lens epithelium, were unaffected.

The third experiment (Exp. 81) was similar to the first except that the flint screen allowed waves down to 305 $\mu\mu$ to pass and that the water cell did not leak. The exposed corneal area was clear immediately after the exposure, but 20 minutes later was found to be distinctly hazy. The epithelium at no time stained with fluorescine. On microscopic examination of the eye, enucleated three days after the exposure, the corneal stroma was found to be swollen in a rather sharply defined area. The epithelium was normal. The corneal corpuscles in the middle third of the cornea showed active proliferation, but in the posterior third had for the most part disappeared. The endothelium was absent behind the exposed area. The iris and lens epithelium were normal.

In the fourth experiment (Exp. 84) a flint screen transparent to waves down to 310 $\mu\mu$ was used and the exposure was one and one half hours. The exposed corneal area was found to be hazy within one hour after the exposure. On microscopic examination of the eye, enucleated four days after the exposure, the corneal stroma was found very slightly swollen and to stain less strongly in eosin in its posterior layers. The corneal corpuscles showed marked proliferation in the posterior portion of the stroma and the endothelium was absent behind the exposed area. The iris and lens epithelium were normal.

In the fifth experiment (Exp. 90) a flint screen (315 $\mu\mu$) was used and the conditions were the same as in the first experiment with the important differences that the water cell was omitted and the exposure was only 30 minutes. Distinct haziness of the cornea was observed within 20 minutes and within 24 hours became very marked. On microscopic examination (48 hours) the cornea showed changes similar to and almost as marked as those of Experiment 88. The corneal epithelium and lens epithelium were unaffected.

Combined Thermic and Abiotic Effects of Radiant Energy on the Cornea.

In four other experiments in which the exposures were prolonged, both abiotic and heat effects were obtained in the cornea. The screens used were transparent to waves less than 305 $\mu\mu$ to 298 $\mu\mu$ in length and the exposures were from one to one and a half hours. In two of the experiments (Exps. 78 and 79) abiotic effects were indicated by loss of corneal epithelium and characteristic changes in the

lens epithelium, and heat effects by the haziness of the cornea occurring within 30 minutes after the exposure as well as by the slightness of the conjunctival reaction. Combined effects were also shown by the microscopic examinations, the corneal corpuscles being completely invisible in the posterior layers of the cornea, but present and showing characteristic abiotic effects in the anterior layers.

The other two experiments were of especial interest because they showed the limit in respect to wave length beyond which we were unable to obtain abiotic effects. The same screen 305 $\mu\mu$ was used as in Exp. 81 just referred to, in which only heat effects were obtained, but the exposures were longer. In Exp. 82 the exposure was $1\frac{1}{2}$ hours and haziness of the cornea was noted within 30 minutes afterwards. The lens epithelium was unaffected, and the only evidence of abiotic action was a slight loss of corneal epithelium occurring after 24 hours. The heat effects on the other hand, were very marked. The corneal endothelium was destroyed in the exposed region and only an occasional corpuscle could be seen even in the anterior layers of the stroma, so that any possible abiotic effects on the corneal corpuscles were masked by the heat effects. At the periphery of the exposed area the corpuscles were in active proliferation at the end of 48 hours. In Exp. 83 the conditions were the same except that the light was focussed upon the surface of the lens instead of upon the cornea, and that the exposure was interrupted for an hour at the end of the first 30 minutes. Haziness of the cornea was noted within 50 minutes after the first exposure. Following the second exposure there was no loss of corneal epithelium and the only evidence of abiotic action was a slight effect on the lens epithelium. The cells were slightly swollen in a small area and a few of them contained characteristic granules. The heat effect on the cornea was about the same as in the preceding experiment.

According to the foregoing experiments with the double lens system, after an exposure of $1\frac{1}{3}$ hours through a water cell and flint screen (315 $\mu\mu$) no changes are produced; after an hour's exposure through a screen (310 $\mu\mu$), slight heat changes; through screen (305 $\mu\mu$) marked heat changes; and through still more transparent screens, marked heated changes combined with abiotic effects. With the flint screen (315 $\mu\mu$) but without a water cell, marked heat changes are produced after 30 minutes exposure, the heat effect of the short waves here being reenforced by infra red waves. It is evident from these results that the specific absorption of the cornea with respect to wave length does not end abruptly, but gradually diminishes from 295 $\mu\mu$ to some-

what beyond 315 $\mu\mu$. It is also evident that the energy absorbed from waves of 305 $\mu\mu$ or over in length, is converted almost exclusively into heat, only the slightest traces of abiotic action being obtained with waves of 305 $\mu\mu$ in length after the most intense and prolonged exposures.

It will be seen that the abiotic effects and heat effects of radiant energy upon the tissues are essentially different. In the case of heat, a certain critical temperature is required before any effect is produced. This is shown by the sharp transition from normal into injured corneal corpuscles at the periphery of the exposed area, and also by the fact that the epithelium, being kept cool by contact with the air, remains unaffected. The heat effect therefore does not vary in direct ratio with the intensity of exposure, obviously due to the fact that dissipation of heat enters into the equation. In the case of abiotic action on the other hand the effect varies directly with the intensity of the exposure. Heat of an intensity just below that sufficient to cause cell destruction, causes cell proliferation. Abiotic action does not directly cause cell proliferation no matter how intense or how slight the exposure. Lastly, heat does not produce the eosinophilic and basophilic granules in the cytoplasm that are produced by exposure to abiotic radiation.

On the other hand, while it is evident that heat does not produce effects similar to those produced by moderate exposures to abiotic waves, extreme exposures to the latter may produce effects not unlike the severe effects of heat. Thus we have shown that severe exposures to waves shorter than 295 $\mu\mu$ in length may lead to complete disappearance of the corneal corpuscles and marked swelling of the corneal stroma. In the case of heat, however, the posterior layers of the cornea are more affected than the anterior layers while in the case of abiotic action the reverse is true.

Thermic Effects of Radiant Energy upon the Iris and Lens.

In Experiment 97 in which the eye was exposed for one minute through a uviol screen to sunlight concentrated by the large mirror, the pigmented iris was severely burned in the exposed area, showing complete hyaline necrosis. The lens epithelium examined, after 48 hours, was unaffected in the pupillary area, but beneath the pupillary

margin it showed an incomplete ring which under the low power of the microscope resembled the wall produced in other experiments by abiotic radiations. Examination under a higher power however, showed that the appearance was due chiefly to the fact that the cells were here in a state of active proliferation, almost every cell being in some stage of mitosis. It was evident that the heat from the pigment layer of the iris, where the latter was in contact with the lens capsule, had stimulated the cells of the latter to proliferation. It is noteworthy that in Experiment 99 in which the exposure was $1\frac{1}{2}$ minutes but in which the iris was unpigmented, neither the iris or lens capsule was affected.

In none of our experiments was the lens injured by the heat generated by the stoppage of rays within its own substance. That clouding of the lens can be so produced, however, even by visible rays alone, with sufficient intensity and prolonged exposure, has already been demonstrated by Czerny [85] and Deutschman [89] in the case of sunlight, and by Herzog [176] who used the carbon arc and suitable filters.

The iris in no other experiment showed heat effects comparable to those just described. In most of the experiments with the magnetite arc and double lens system the iris was not greatly exposed to the light owing to the artificial mydriasis, but in Experiment 88 in which the most intense heat effect was obtained in the cornea, the iris showed hemorrhages near the pupil. In Experiments 83 and 90 the iris became greatly contracted towards the end of the exposures, and remained so for several hours, but again dilated within 24 hours.

Thermic Effects of Radiant Energy upon the Retina.

In a number of our experiments, some of which were made with other purposes in view, we obtained heat effects in the retina in spite of an interposed water cell 5 cm. thick. They were obtained mainly in two ways, one by the use of sunlight reflected from a silvered glass concave mirror 26 cm. in diameter and 1.5 meters in focal length, and the other by the use of the magnetite arc light concentrated by the single quartz lens system. A full description of the mirror and the calculated energy derived from it is given on page 721. The calculated energy on the retina given by the quartz single lens system is given on page 724. The burns were obtained through screens that obstructed all waves less than 335 $\mu\mu$ in length as well as through

more transparent screens. In the case of sunlight the exposures were from one-fourth second to one and one-half minutes, and the resulting burns were always severe, the retinal tissue being actually coagulated as will be described. In the case of the magnetite arc the exposures were from ten minutes to one hour and the burns were much less severe. In addition to these, heat effects involving the pigment epithelium alone were obtained in two experiments with the quartz double lens system (Exps. 88 and 89) in each of which a large area of the fundus was illuminated. One of these was in the case of an aphakic eye, and the other in a case of exposure without a water filter. The significance of these experiments in connection with the questions of eclipse blindness and allied phenomena is discussed elsewhere (page 720).

That the severe effects produced by concentrated sunlight were due to heat was obvious from their histological appearances and from the fact that the light intensity at the focus was found in all cases to be sufficient quickly to ignite a match or piece of paper. That the relatively slight effects produced by the magnetite arc were also due to heat, was obvious from the fact that only the pigment epithelium and outer retinal layers were affected and sometimes the pigment layer alone. If the effects had been due to the abiotic action of light the inner nuclear layer and ganglion cells would necessarily have been equally or even more greatly affected. Moreover, as we have already shown, when the corneal epithelium and lens epithelium were exposed to light of greater intensity and shorter wave lengths than was the retina in these experiments, and for a much longer time, no changes were produced in them. Thus in Experiment 53 a heat effect in the retina was obtained after 12 minutes exposure to light passing through the lens of the eye, that is, to waves longer than 330 $\mu\mu$, whereas in Experiment 85 no effect was produced on the cornea after an exposure of $1\frac{2}{3}$ hours to light of greater intensity containing wave lengths as short as 315 $\mu\mu$. This is easily explicable on the assumption that the retinal changes under consideration were due to heat, since the cornea and lens must each absorb a far less proportion of visible and infra red rays that reach them than does the pigment epithelium of the retina. On the other hand it is absolutely inexplicable on the assumption that the retinal changes were due to abiotic action, since it is inconceivable that the corneal and lens epithelium would be unaffected by abiotic action of light sufficient to produce nuclear fragmentation in the outer muclear layer and pigment epithelium. The character of the histological changes clearly indicates that the heat

conversion took place chiefly in the pigment epithelium and inner layers of the chorioid, and that the outer layers of the retina proper were affected by the heat conducted therefrom.

An interesting problem is the exact determination, under various conditions, of the minimum intensity and duration of exposure to visible and infra red rays necessary to produce heat effects on the retina. A discussion of this problem will be found on pages 721 and 732.

The experiments in which heat effects on the retina were obtained by means of the magnetite arc were Experiments 53, 55, 57 58, 59, 88, 89.

Following are the experiments with sunlight concentrated by the large mirror.

Experiments.

Sunlight Focussed on Cornea by Large Mirror.

Experiment 95. Without water cell or screen. Pigmented eye. Three exposures, $\frac{1}{4}$ second, $\frac{1}{2}$ second, and 10 seconds respectively. No inflammatory reaction. Enucleation at end of 33 days. Lens epithelium normal. Three burned areas in fundus of different grades of severity.

Experiment 96. Water cell. Flint glass screen (335 $\mu\mu$). Albino. Exposed 4 seconds. No inflammatory reaction. Lid reflex to light abolished. Enucleation at end of 48 hours. Two contiguous burned areas in fundus, one exactly on disc. Marked hemorrhagic retinitis. Slight hemorrhage from retina into vitreous.

Experiment 97. Blue uviol screen. No water cell. Pigmented eye. Exposed 1 minute. After 1 hour: Pupil contracted to $\frac{1}{2}$ normal size, does not react to light. After 24 hours: Lid margins inflamed, lower one ulcerated, no lid reflex to light. Cornea clear. After 3 days: Cornea shows purulent infiltrate below (infected from ulcerated lid). Enucleation. Fundus shows two contiguous burned areas, one at margin of disc. Microscopic examination: (3 days): Slight purulent infiltration of cornea. Hyaline necrosis of iris. Lens epithelium normal in pupillary area, shows proliferative changes beneath pupillary margin (heat effect due to contact with heated iris. See page 696).

Experiment 98 (Pl. 4, Fig. 14). Water cell. No screen. Albino. Exposed 14 seconds (misty day). No inflammatory reac-

tion. Enucleation at end of 6 days. Fundus shows burned area just beneath optic disc. Lens epithelium normal.

Experiment 99. Blue uviol screen. Water cell. Albino. Exposed $1\frac{1}{2}$ minutes. No inflammatory reaction. Enucleation at end of 12 days. Fundus shows burned area undergoing repair. Cornea, lens epithelium, and iris normal.

Experiment 100. Blue uviol screen. Water cell. Albino. Total exposure, 10 minutes, one second on, one second off. No inflammatory reaction. Enucleation at end of 7 days. Retina shows no burned areas. Cornea, lens epithelium, and iris, normal.

Experiment 101. Blue uviol screen. No water cell. Pigmented eye. 220 exposures, $\frac{1}{2}$ second each, with intervals of 1 to 3 seconds. No inflammatory reaction. Fundus normal.

Character of the Thermic Effects Produced in the Retina.

In the experiments in which the pigment epithelium alone was affected no changes were noted macroscopically. In most of the other experiments the lesions could be seen with the ophthalmoscope or better still on opening the eye after enucleation. They appeared as sharply defined reddened spots. Some of those obtained after exposure to sunlight showed blood extending from them into the vitreous humor. Some of the spots were observed only after the eye was placed in Zenker's fluid. In Experiment 96 in which a burned area involved the optic disc, there was intense hemorrhagic retinitis apparently due to thrombosis of the central vein. The spots produced by sunlight measured about 2.5 mm. in diameter. Those produced by the magnetite arc and single lens system were about 3 mm. in diameter as measured under the microscope with reference to the effects on the pigment epithelium, but only about 1 mm. in diameter as measured with reference to the effects on the retina proper when this was involved. This concentration of the effects in the center of the area was no doubt due to two facts, one being that the light was actually more intense here, and the other that towards the periphery of the area the heat generated in the pigment epithelium became rapidly dissipated.

Microscopical: The most striking feature of all the burned areas whether due to long or short exposures was their sharp demarcation, illustrating again here as in the case of the cornea how sharply critical

is the temperature required to injure tissues. In all cases the pigment epithelium was the most severely affected of any portion of the retina, and in the slightest burns it alone was affected. This was true also in albinotic rabbits in which the epithelium was free from pigment. The other structures were affected in the following order according to the intensity of the action, the rods and cones, the chorio-capillaris, the outer nuclear layer. The inner nuclear layer, the ganglion cells, and nerve fibre layer, were affected only after extremely intense exposures and in our experiments were not affected after exposures to the magnetite arc but only when concentrated sunlight was used.

The slightest change that can be definitely made out in the pigment epithelium 48 hours after exposure consists in the cytoplasm of a greater or less number of cells staining intensely in eosin. When the effect is somewhat greater, vacuoles appear and may be so large and numerous that the cells appear almost completely transparent, the cytoplasm showing a delicate reticulum with the minute nodes at the junction points. In case the epithelium is pigmented the pigment appears to be separated from the membrane of Bruch by large vacuoles, and the nucleus may show marked pycknosis. When the effect is still greater the cell reticulum completely disappears leaving only the nucleus in the clear space thus formed. The nucleus may show fragmentation or simply chromatolysis. The basophilic and eosinophilic granules characteristic of abiotic action are not seen. In the somewhat more severe burns the pigment cells entirely disappear leaving only the pigment. In one eye examined six days after exposure the injured epithelium is found replaced by epithelium which has evidently grown in from the periphery, but between the new layer and the rods and cones numerous swollen vaculated and otherwise altered pigment cells remain. The changes in the pigment epithelium are best seen in plane section, and to determine the character of the slightest changes it is important to compare the appearances seen in the exposed eye with those of a normal eye.

Forty-eight hours after exposures sufficient to affect the rods and cones, the outer limbs of the latter are found to be broken up into coarse granules, while the inner limbs are swollen to large bladder-like structures each containing a few fine granules. There may also be a greater or less number of red blood corpuscles among the rods and cones due to diapedesis from the chorio-capillaris, and also a certain amount of serum. When the outer nuclear layer is affected, the nuclei lose their peculiar cross striations, becoming intensely pycknotic and some of them undergoing fragmentation. With this degree of

injury the pigment cells have disappeared, and fragmented nuclei can sometimes be seen in the inner layers of the chorioid. The inner layers of the retina, including the ganglion cells, remain normal in appearance. (Pl. 4, Fig. 12.)

In the experiments in which sunlight was used, as already noted, much more intense heat effects are found in the retina and chorioid. Here the inner layers of the retina are disintegrated while the outer layers appear intact. This is undoubtedly due to the fact that the latter have been coagulated and thus fixed by the heat. At the periphery of the area the appearance is reversed, the inner layers being normal while the outer layers show the less marked changes already described.

Two to six days after such an exposure the coagulated rods and cones maintain their normal appearance except that they stain abnormally deeply in eosin. The nuclei of the external nuclear layer are likewise coagulated, but have lost their cross striations and stain more deeply than normal. The internal nuclear layer shows different appearances in different places evidently according to the heat intensity. In some places the nuclei still take the basic stain and show fragmentation. In others they stain in eosin and are not disintegrated, while in still others they have entirely disappeared. The nerve fibre layer is completely disintegrated and the ganglion cells have entirely disappeared or stain only in eosin. At the periphery, the ganglion cells with their Nissl bodies stain less and less in thionin as the burned area is approached until they become entirely eosinophilic. The inner surface of the retina is in some cases coated with a thick layer of fibrin. The pigment epithelium is coagulated and retains its normal position in the exposed area. At the periphery it is disintegrated. The chorioid behind it shows large extravastions of blood and marked nuclear fragmentation. There is no cellular infiltration, purulent or otherwise, of either the chorioid or retina. (Pl. 4, Fig. 14.)

After two months the retina is found replaced by neuroglia containing migrated pigment cells. In some cases the chorioid is apparently normal and the pigment epithelium reformed. In others the latter is absent and the chorioid replaced by two or three layers of a vascular fibrous tissue.

Theory of Action of Radiant Energy on the Tissues.

A useful conception of the effects of radiant energy upon the tissues of the body is that the heat effect is due to increased molecular motion while the abiotic effect is due to direct atomic disintegration of the molecules with immediately resulting chemical changes. The first effect of the increased molecular motion is to produce a physical change analogous, for example, to the melting of ice. When the motion reaches a certain critical rate the molecules are broken up and various chemical changes result. Both heat effects and abiotic effects theoretically may be produced by rays of any wave length, but practically in the case of short waves the heat effect is generally negligible, while in the case of long waves the abiotic effect is negligible. Our experiments show that for human cells the abiotic effect becomes negligible within a very short range of wave lengths, that is between 305 and 310 $\mu\mu$. For bacteria it becomes negligible still sooner, at less than 295 $\mu\mu$. Under ordinary conditions heat effects are also negligible here, and in fact all through the visible spectrum, although with extreme intensities such as afforded by concentrated sunlight they may be produced, as in eclipse blindness, for instance. It is in reality due to the fact that abiotic effects and heat effects are negligible in the region of the spectrum indicated, that sunlight under usual conditions is not destructive to human life. This fact, considered from the standpoint of evolution, suggests a relation of light to the origin and structure of living matter, but a discussion of this aspect of the subject would lead too far.

Since according to this conception the abiotic action of light is directly upon the structure of the molecules, slight chemical changes are produced after almost infinitesimal exposures. Theoretically, of course, there is a limit of exposure below which no disintegrating effect is produced upon the molecules, so that a series of such short exposures would produce no summative effect. Practically, however, this would be impossible to demonstrate in the case of living cells. On the other hand, in the case of living cells summation of the effects of a series of exposures, if the intervals were too long, would not accurately occur, since the repair of the injury would take place to a greater or less extent. Thus we have found in the case of the corneal cells that summation of effects becomes much less exact when the intervals of exposure are over twenty-four hours.

In the case of heat, deleterious effects on the tissues must likewise be due to chemical changes. Since, however, these changes take place only when the molecules have reached a certain rate of motion, under ordinary conditions a measurable time interval must elapse before they begin. The length of the time interval depends upon the intensity of the light and upon the rapidity with which dissipation of heat occurs, and thus varies greatly under different conditions. Under ordinary conditions, however, the time interval is of considerable length, so that a series of exposures does not produce a total effect equal to that of a continuous exposure of the same total length, and may not produce any effect at all. From a practical standpoint therefore this fact constitutes a fundamental difference between the abiotic effects and the heat effects of radiant energy.

Unless light rays are absorbed by substances they can of course produce no effect upon them. Thus, as we have shown, waves over 295 $\mu\mu$ in length unless extremely intense have no effect on the corneal stroma which is relatively transparent to them but have a markedly deleterious effect on the corneal corpuscles which absorb them. It does not necessarily follow, however, that because light rays fail to pass through a given substance they must produce an effect upon it. For they may simply be changed into light waves of longer wave length (fluorescence) or their energy dissipated in the form of heat of an intensity too low to produce any changes. Both of these transformations must take place in the case of the lens of the eye since light waves are constantly being stopped in it. That fluorescence actually occurs in the lens is, of course, well known and easily demonstrated.

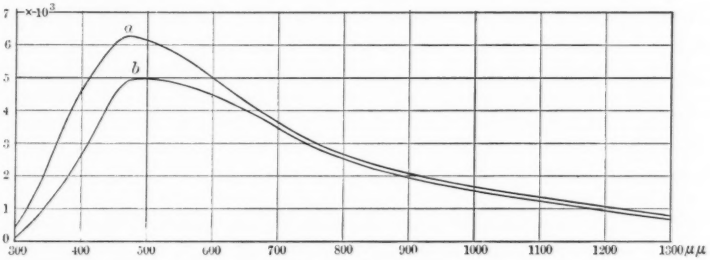
Assuming that the abiotic action of light of given wave lengths upon protoplasm is directly proportional to the coefficient of absorption of the protoplasm for, that wave length, Henri [163] and his wife have determined this coefficient for egg albumin and a large number of waves. The curve plotted from their results given elsewhere (page 645) shows that the absorption becomes practically nil at and near 310 $\mu\mu$, so that the abiotic action must be very slight here. Moreover, since this method does not allow for the fact that the absorbed rays produce heat as well as abiotic effects, the abiotic action is undoubtedly less than is indicated by the curve of absorption. These results, therefore, confirm in a striking manner those obtained by us by actual experiments on the cornea.

The preceding discussion has concerned mainly the direct effects of light upon the molecules of the tissues without reference to histological and clinical manifestations. The latter are of course, too

complicated for complete analysis since fully to understand them would require a knowledge not only of the chemical changes originally produced, but of the vital processes of the cells. An interesting question in regard to them is that concerning latency of their appearance. In the case of abiotic action, as has been pointed out, absolutely no visible change either histological or clinical takes place immediately after the exposure, and usually not for several hours. This is no doubt due to the fact that time is required for the chemical changes to produce physical alterations. In the case of heat effects, it is a matter of common experience that latency also occurs and that the time interval varies with the intensity of the exposure, but it is a far less striking phenomenon than in the case of abiotic effects. This may be due, among other causes, to the fact that the energy required to produce chemical changes by heat is so great owing to the rapid dissipation of the latter, that under ordinary conditions the critical point is quickly passed and an excessive effect produced.

Abiotic Energy in the Solar Spectrum.

As has already been noted the solar spectrum when filtered through a thick layer of atmosphere as at sea level when the sun is low fades out at about 305 $\mu\mu$. At high altitudes and with the sun running



a. Mt. Whitney z. d. = 0°
b. Mt. Whitney z. d. = 60°

Figure 5. Distribution of energy in solar spectrum.

high, it extends to about 295 $\mu\mu$. Under extremely favorable conditions some very faint traces of the spectrum were registered by Cornu [79] down nearly to 292 $\mu\mu$.

But substantially the whole of the solar spectrum which is capable of producing abiotic action lies between 295 $\mu\mu$ and 305 $\mu\mu$, is evanescent under most conditions, and only possesses pathological significance at high altitudes and especially in extreme cold. There is good reason to believe that the atmosphere is considerably more permeable to ultra violet radiations at low temperatures than under ordinary conditions, particularly as regards the extreme radiations. Figure 5 shows from the data of Abbot the distribution of energy in the solar spectrum in curve (a) at Mt. Whitney for a zenith distance of 0°, in curve (b) also at Mt. Whitney (14,000 ft.) but for zenith distance 60°. Near the latter limit lies the general range of solar radiation as observed at the surface. Two things in these curves are particularly noteworthy, first that in both and especially at the higher altitude the maximum radiation and indeed the bulk of the radiation in general lies within the visible spectrum. Second, the maximum energy lies not in the red, but in the case of the high altitude energy fairly in the blue at about wave length 470 $\mu\mu$ and at the lower altitude in the green at wave length about 500 $\mu\mu$. So far as the solar spectrum is concerned, therefore, the heat energy is chiefly within the visible spectrum. No distinction therefore can be drawn between the visible and the infra red spectrum on the ground of heat radiation and all attempts to separate thermic effects by cutting out the visible spectrum are therefore futile. So long as this reaches the eye it carries with it the solar heat in its greatest intensity. From the area of the curves here shown it appears that of the energy at high altitudes only a very small proportion, of the order of magnitude of one quarter of 1% lies within the region 295 to 305 $\mu\mu$. Even this small quantity is evanescent at the sea level and at ordinary temperatures. It is to the small remaining trace of abiotic rays here noted that the phenomena of snow blindness are due. From the clinical standpoint snow blindness is found to occur only as a photophthalmia of relatively very mild degree and under exposures usually for a long period and either at very high altitudes or very low temperatures or with both these conditions concurring. On snow fields the exposure of the eye to solar radiation, ordinarily greatly ameliorated by the obliquity of the incidence, is rendered much more severe by the reflection from the snow which is a good reflector down to the extreme ultra violet of the solar spectrum. One would not go far wrong in estimating that the radiation reaching the eye under such circumstances is of the order of magnitude of a million ergs per square cm. per second. A single square meter of snow at 2 meters distance would reflect to the eye

almost a tenth of this amount with the altitude and sun favorable. Assuming now that one quarter of 1% of this quantity, that is 250 ergs per square cm. per second is within the abiotic region 295 to 305 $\mu\mu$ it is easy roughly to determine the exposure which is likely to produce snow blindness. We have already seen that a well marked photophthalmia can be set up by a radiation in abiotic rays of about 2,000,000 erg seconds per square cm.

Now assuming that of the total radiation which would be received direct, half, through direct and reflected action, reaches the eye of one traveling among the high snow fields. The energy in total abiotic radiations would be about 1250 ergs per square cm. per second. If all of this quantity had the average abiotic effect on the conjunctiva and the cornea a little less than 27 minutes exposure would be required to make up the 2,000,000 ergs seconds just referred to and to produce symptoms of photophthalmia. As a matter of fact the region from 305 $\mu\mu$ to 295 $\mu\mu$ has much less than the average abiotic effect. Our crown glass screen # 7 cuts off the ultraviolet at 295 $\mu\mu$ substantially just at the end of the solar spectrum. Experiments made with this screen on the magnetite arc which is fairly strong from 295 $\mu\mu$ to 305 $\mu\mu$ showed that this screen increased the exposure necessary to produce photophthalmia eighteen times. It therefore appears that at a high altitude in the snow fields an exposure of 7 to 9 hours under extreme conditions would be required to produce photophthalmia as severe as that which we have here recorded as typical, *i. e.*, involving stippling of the corneal epithelium. Clinically snow blindness very rarely reaches this phase, since, although the exposures may be long the intensity of abiotic solar radiations reaching the eye would be seldom as great as the maximum amount just mentioned. For instance Schiess-Gemusens [324] reports two cases of ordinary snow blindness which fell under his own observation in which the ordinary symptoms occurred after practically all day exposures showing very marked conjunctivitis without any visible effect on the cornea. Inasmuch as the exposures in casual climbing on an all day trip are considerably less severe than with steady full exposure to the snow fields, it is fair to assume that this latter condition might produce snow blindness in perhaps half the time previously mentioned. This checks well with our experiment on solar erythema where an exposure of 6 minutes at .5 meter from the magnetite arc unshielded gave a slight but definite erythema of the skin. At sea level and under ordinary circumstances the critical exposure for snow blindness would undoubtedly run to many hours. It is well

known clinically that snow blindness has often been reported in polar exploration. In high latitudes at sea level the abiotic energy is greatly reduced but three circumstances enter the case to increase the danger of snow blindness. First the hours of sunlight are very long, second, intense cold is believed to decrease the atmospheric absorption for the extreme ultra violet, and third, the exposure of the eye to prolonged and intense cold, while it may not actually lower the vitality of the cells to render them more easily attacked by abiotic radiation, unquestionably would tend to lower their recuperative power and so effect the summation of exposures which ordinarily would be relieved by continuous repair.

Solar Erythema.

These data on solar energy at once call up the question of solar erythema generally attributed to the effect of ultra violet radiation.* Clinically this bears a suggestive resemblance to photophthalmia in that it has a period of latency and a similar period of duration. Further it is well known to occur easily at high altitudes with the sun running high, that is under circumstances which afford a fair amount of abiotic rays. The best recent investigation of this matter is that by Dr. deLaroquette,[220] Surgeon Major of the French Army in Algiers. His experiments under the intense tropical sun show the connection of solar erythema with the abiotic rays very clearly. In the first place in most cases he noted a primary erythema clearly due to temperature and perhaps associated with heated air as well as radiation, occurring only when the temperature is 30 degrees C. or more. This is followed after a period of latency of an hour or two by a secondary photochemical erythema going on under severe exposures to hemorrhagic pigmentation, local oedema and subsequent desquamation. Experiments in exposure of the skin under screens showed under layers of quartz and water, both of which are highly transparent to abiotic rays, the secondary erythema was well marked.

* It may be mentioned here that there are certain rare chronic affections of the skin, notably *xeroderma pigmentosa*, that are believed to be due to exposure to day light, chiefly because they involve only the exposed surfaces of the body. It is supposed that for some unknown reason the skin is in such cases abnormally sensitive to abiotic radiations. Possibly other slight irritants applied for the same length of time would produce similar effects. Two cases have been recorded in which the cornea was involved, and one of us has personally examined such a case.

Under window glass and violet or blue glass it was slight, even after considerable exposures, while with yellow, red, green or black glass it was absent, although in each case the primary erythema was marked. No investigation was made of the absorption of the various glasses, but from our experiments the clear, the blue and the violet glasses are likely to let through the margin of the abiotic radiations in the thickness, 2 mm., here employed. Yellow, red, green and black glasses would certainly cut these off. The skin in open exposure to sunshine is very much more exposed to the full energy of the solar radiations than is the surface of the cornea and conjunctiva and for abiotic effects on the skin practically the full strength of solar radiation is available. One would therefore expect to get action from the abiotic rays in, at most, half the time noted with respect to the cornea and conjunctiva for a similar degree of effect. In other words, one should get in a couple of hours well marked effects and undoubtedly slight erythema in an hour or so, as experience well shows is the case, assuming somewhat similar degree of sensitiveness in the epithelial cells. In case of extreme exposure to heat radiation distinct heat effects may be found in either case. Dr. deLaroquette's observations on the human skin were fully checked by exposures on shaven areas on the skin of a guinea pig showing the same general phenomena. Dr. deLaroquette also suggests that low temperature and wind drying the epidermis and provoking intense superficial vaso-constriction tends to exaggerate solar erythema. In this way some rational account can be given of its occurrence under conditions of cold and severe wind alone when the abiotic action of the solar radiation would be small or even wanting, in which case the effect would be a primary rather than a secondary one. Finally, in solar erythema, as in photophthalmia, repeated exposures of somewhat subnormal intensity give acquired tolerance, while the skin is, as well known, somewhat hypersensitive to severe exposures following each other without time for the lesions to undergo repair.

Our experiments with the bare magnetite arc as source indicate that the liminal exposure for perceptible abiotic effects is practically the same for the more sensitive parts of the skin as for the conjunctiva. The inner portion of the forearm was the portion of the body exposed in our work on liminal exposures. Here with 6 minutes at .5 meter, which corresponds very well with the production of mild photophthalmia, a slight reddening of the skin appeared some few hours after exposure, rose to its maximum inside of the first 24 hours and vanished within a day or two leaving no trace. Through the double

lens system and crown glass screen (295 $\mu\mu$) already described, the liminal exposure was between 15 and 30 seconds, the former figure giving no traces and the latter slightly more than a liminal exposure. In all exposures over half a minute there was immediate heat erythema and a subsequent development after a period of latency of a few hours. There was a distinct but slight feeling of heat during the exposure and a rather rapid extension of the erythema somewhat beyond the limits of the 5 mm. stop which limited the area exposed. In cases of severe exposure to the sun we are inclined to think that this primary erythema due purely to the effects of heat is of considerable importance in the total results experienced. We found, as did Dr. deLaroquette, that vaseline acted as a fairly complete preventive as regards both primary and secondary erythema, particularly the latter, while glycerine gave a slight protective action in our results, more than would seem to be warranted in view merely of its transparency to abiotic rays. From these observations and from the clinical facts, often showing erythema greatly disproportionate to the intensity of abiotic radiation likely to be present, it seems probable that ordinary sunburn is due to a mixture of thermic and abiotic effects of which the former are often the more prominent, although they generally cannot readily be separated from the secondary abiotic effects, the development of which they tend to mask.

Erythropsia.

So-called erythropsia is the name of a phenomenon rather than of a pathological condition. The clinical records are numerous but vague. They all indicate a condition, generally very temporary, in which the patient finds a more or less ruddy tinge in everything seen. There is nothing definite in the tint of the coloration or the period through which it is observable. It apparently runs from various shades of orange and rose to a fairly full red. The most definite description given of the apparent color, which evidently pertains to a rather extreme incidence, is given by Fuchs [126], who compares it to a strong fuchsin solution with a trace of eosin solution. A cursory view of the clinical records indicates that the cases cited fall into three general divisions. First, cases associated with neurosis such as those given by Charcot and others (cited by Wyeller [422]). These clearly cannot be associated with any pathological condition of the visual

apparatus. Second, there are many recorded instances of traumatic erythropsia some of which at least evidently are associated with the actual infiltration of the eye media with blood. Third, one finds a vast majority of instances which one may term photo-erythropsia in which the observed appearances, one can hardly dignify them by the name of symptoms, are associated with over exposure to light. These are so entirely without pathological significance that we should hardly consider them here save for the fact that the phenomena have been by some writers like Widmark [411], Fuchs [126], and others, subsequently attributed to the effect of ultra violet radiations. As this erroneous conception of the fact still persists in spite of the admirable work of Vogt,[402] it is desirable here to note the relation of this so-called erythropsia to the general phenomenon of color vision. The whole subject was thoroughly investigated recently by Wydler [422] who very plainly showed that erythropsia is due to the red phase of the negative after image following over-exposure to light, ordinarily brilliant white light, although green and blue green illumination is even more effective. The association of the phenomenon with ultra violet radiation appears to be due to the fact that photo-erythropsia has been often observed after the intense glare which produces snow blindness and not infrequently in the aphakic eye after an operation for cataract. That the ultra violet really has nothing to do with the matter is clearly shown by Vogt [402] who found that erythropsia could not be produced experimentally by the ultra violet rays alone, but very easily by light rays containing no ultra violet. We need only add here that it is possible to produce marked erythropsia through euphos glass which transmits no ultra violet, through B-naphthol-disulphonic acid which also cuts off the ultra violet, and through dense flint glasses which eliminate all of the ultra violet which could with any certainty get through the lens. Also it is produced with great facility by radiation through green and blue green media which intercept the ultra violet completely, but flood the eye with light of a color certain to produce a strong red phase in the after image. The truth seems to be that the so-called photoerythropsia is merely the result of such unequal fatigue of the primary color sensations as leaves for a greater or less time thereafter a color sensation predominantly red. This conception clears up at once the difficulty of accounting for the partial erythropsia which has been noted by Purtscher [287] and others, since in cases of unequal exposure of various parts of the retina to brilliant light the fatigue effects necessarily must vary over the field of vision. A glance at figure (6) will render the situation clear. The curves in this figure

are those of the three normal color sensations reduced to equal areas as determined by Exner. If any of these primary sensations are fatigued that of the remaining color or colors becomes the predominant tint seen. This has been beautifully shown by Burch who by suitable means fatigued to complete exhaustion each one of the sensations and various combinations of them. Burch [61] found that of the three the red first regained its sensibility, after perhaps ten minutes, followed by the green and last of all by the blue where marked fatigue might persist for several hours. Red vision is therefore normally to be expected after fatigue of all three sensations since the red recovers first and in case the green and blue are more fatigued than the red the latter will be more notably predominant. As the maximum luminosity of the spectrum lies in the green and as at high altitudes under a clear sky the blue is relatively strong, exposure in the

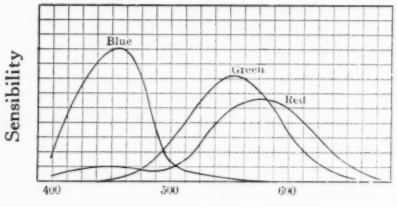


FIGURE 6. Primary color sensations, after Exner.

high snow fields necessarily fatigues these two sensations predominantly, and photoerythropsia in greater or less degree may reasonably be expected.

As a corollary we may note the reputed activity of the quartz mercury arc in producing erythropsia. On figure 2, Plate 5, marked in their proper positions are the three chief lines of the mercury spectrum at waves lengths 454 $\mu\mu$, 546 $\mu\mu$, and the pair at 578 $\mu\mu$. It will be seen that they lie in positions which indicate stronger fatigue of the green than of the red and marked fatigue in the blue. The green line at 546 $\mu\mu$ is by far the strongest of the three followed by the yellow pair at 578 $\mu\mu$ and by the strong line of blue. The chief red line in the spectrum is relatively very weak hence fatigue weakens the green most, red in the next degree and blue relatively little. After fatigue,

therefore, the red comes easily and quickly into activity and unites with the residual blue to produce very marked erythropsia of a distinctly rosy tinge. This lasts for some minutes, while traces of the disturbance of the blue and green vision may still be found after prolonged exposure for a period of several hours, and this like other forms of color fatigue takes place whenever the light which affects the three primary sensations has been active, quite irrespective of whether the ultra violet is present or absent.

As to the aphakic eye Wydler [422] has noted the probable effect of coloration of the lens in this connection. When an eye has been shielded, often for years, against any strong access of the blue and violet and against the extreme end of the green sensation as well and is then after recovery from cataract operation exposed to strong daylight, it is merely a phenomenon to be expected if there is predominant red vision after fatigue. It would not be surprising, for that matter, if after long disuse of the color sensations toward the blue end of the spectrum fatigue were easier and recovery less prompt than in the normal eye. Aphakic patients in whom the pupil is often greatly enlarged as a result of iridectomy are likely to receive extraordinarily intense illumination on the retina, and hence may show the phenomenon of color fatigue to an exceptionally great extent. Exposed in the open the color fatigue is very marked, and when this wears off after the exposure ceases, the return of the red sensation may very naturally be accompanied by some degree of erythropsia for this reason alone. One of us has recently examined a case in which erythropsia so produced was a characterisitc condition.

Vernal Catarrh.

Spring catarrh is an uncommon disease of the conjunctiva that most often affects the upper lid, much less often the bulbar conjunctiva around the corneal limbus, and almost never the two together. It is extremely chronic, lasting from 3 to 20 years, and is associated with the formation of peculiar granulation tissue, infiltrated with eosinophilic leucocytes to an unusual degree, containing downgrowths of epithelium from the surface. In the case of the conjunctiva of the lid, the new tissue forms within the papillae, thus giving rise to large flat papillary growths. The symptoms of irritation, photophobia, lacrimation, and itching, are most marked in the spring and warm

seasons of the year, usually disappearing during the winter months. For this reason sunlight has been suggested as the etiological factor in the disease.

This hypothesis was first advanced by Schiele [323] and advocated by Kreibich [211] who showed that an occlusive bandage had a favorable effect upon the symptoms. This effect, however, may be explained in other ways than by the shutting out of light. Birch-Hirschfeld [34] repeatedly exposed the conjunctiva of a rabbit within a period of 18 months to the "Uviol lampe" of Schott and obtained changes stated to be not unlike those of vernal catarrh. He does not, however, accept the view that the latter is due to ultra violet light. No doubt similar changes could be produced by other irritants frequently applied.

The evidence for the view that vernal catarrh is due to the action of sunlight, therefore amounts to little more than the fact that the symptoms are most pronounced in the spring and summer. This fact, however, is accounted for even better on the more recent theory that the disease is due to pollen. Moreover the following objections, that to us seem insurmountable, may be urged against sunlight as a cause. In the first place if vernal catarrh is due to sunlight the lower lid, which is not only more exposed, but thinner and more transparent, should be more affected than the upper lid, whereas, as a matter of fact, it entirely escapes involvement. In this connection it may be noted that in cases of trachoma, a somewhat similar disease, the lower lid also usually escapes and here the disease is undoubtedly due to some infectious agent. Similarly this theory is inconsistent with the fact that the bulbar conjunctiva, which is directly exposed to the light, is but seldom affected, and almost never affected in association with the palpebral form of the disease.

Finally, the possibility of abiotic action is ruled out by the fact that it is impossible for abiotic waves to pass through the entire thickness of the lid, if only on account of its rich vascularization. This objection does not apply to possible heat effects produced by visible or infrared rays, but in this case it would be necessary to assume exposure of the eyelid to direct sunlight for considerable periods of time as well as special sensitiveness of the conjunctiva to heat, neither of which conditions seems possible.

Senile Cataract.

The theory has been advanced (see page 780) that senile cataract is due to exposure of the lens to daylight, particularly that from the sky. This is based solely on the fact that the cataractous changes usually begin in the lower part of the lens. It is undoubtedly true that the changes do first appear below, but as a rule they are so far below that they are in a portion of the lens completely shaded by the iris. Thus it is most often necessary to produce artificial mydriasis before incipient lens changes can be seen with the ophthalmoscope. Moreover, if the cataract were due to exposure to light, the pupillary area should be the first affected, since from such an extended source as the sky it receives the greatest concentration of light, and since the chief absorption must occur here. We must conclude, therefore, that there is no sound evidence for this theory of cataract formation.

A possible explanation of the fact that the lower part of the lens is usually first affected in senile cataract is that the structure of the lens may normally be slightly different here than elsewhere. From a developmental standpoint this is indicated by the fact that coloboma of the lens usually occurs below. Burge [61a] has recently attempted to supply an experimental basis for the view that ultra violet light is responsible for cataract. He found that the rays from an unscreened quartz mercury vapor lamp had almost no coagulating effect upon the lens protein even after an exposure of 72 hours at a distance of 5 cm. but that when acting in the presence of weak solutions of calcium chloride, sodium silicate, or dextrose, coagulation occurred. Since in senile cataracts calcium, magnesium, and sometimes silicates, are greatly increased, and in diabetic cataracts dextrose is presumably present, Burge assumes that these cataracts are due to the action of ultra violet light. That is, he assumes that these substance are present in undue quantities in the lenses of certain individuals and that this renders their lenses vulnerable to the short waves of daylight.

This assumption is sufficiently controverted by the fact just mentioned that senile cataract usually begins at the periphery below. But in addition, other serious objections to his argument may be pointed out. In the first place, in traumatic cataracts and cataracts due to inflammatory conditions, calcium salts, and no doubt magnesium and other salts, are deposited in great abundance, and the lens may even become completely calcified. In fact, the same thing occurs in dead tissues anywhere in the body, so that the reasonable

assumption is that the presence of these salts in senile cataract is a result not a cause. Then, too, Burge made use of intensities of exposure and wave lengths to which the lens is never subjected during life. The cornea completely screens it from practically all the short waves found effective by him. The longest waves with which he could coagulate proteins were 302 $\mu\mu$ in length and the effect produced by these was insignificant.

Burge suggested also that his results might apply to glassblowers cataract, overlooking the fact that the latter typically begins at the posterior pole of the lens, whereas in his experiments the part of the lens away from the light was little if at all affected. It is of course obvious that the slight loss of transparency he sometimes observed in this part of the lens could not have been due to the direct action of the light, since the effective rays could not have penetrated so far. The fatal objection to Burge's theory as applied to senile cataract is that the ultra violet solar rays cannot reach that portion of the lens where cataract generally begins, and that portion of the lens where ultraviolet light has the best chance of action is affected only at a late stage of development.

Concentration of Energy in Images.

We have already shown that superficial action of radiant energy on the eye depends on the actual energy in ergs per square cm., or other convenient measure, which falls upon the surface. Such value is directly as the energy of the source and inversely as the square of its distance. The density of incidence of energy at points within the eye is obviously dependent on the amount to which the superficial energy is concentrated by the refracting media, and at the retina the concentration of energy is determined by the size of the image and the aperture of the refracting system, which is determined by the area of the pupil. In dealing with an extended source the image is correspondingly extended and the surface density of energy in the image is correspondingly reduced. Hence it is that with sources like the tube of the quartz arc the image density is relatively small, while with point sources or those of very small area, like the electric arc, the retinal area is correspondingly small and of the total energy reaching the pupil there is a greater concentration in the image. Within limits the intensity of the effect on the retina is then directly proportional to the intrinsic brilliancy, or radiation per unit area of the

source. The mere lowering of the intrinsic radiation by spreading out the source therefore greatly lessens any possible effects of energy which may reach the retina, while it does not in any way affect the radiation which may reach the cornea and conjunctiva. There is, however, a very notable limitation to this principle which comes into play in considering small and intense radiants, as was long ago shown by Charpentier [69]. When the image of a luminous object reaches the diameter of approximately 0.15 mm., variations of intrinsic radiation at the source cease to be significant and the apparent intensity of the source varies simply with the inverse square of its distance. This corresponds to the visual angle of about 40 minutes of arc. For areas of greater dimensions one must reckon with the size of the image as determined by the ordinary laws of geometric optics, but for radiants of less than this dimension the image may be taken as of constant area corresponding to the circle of diffusion, and the energy concentrated in it varies as the inverse square of the distance of the radiating source. In any case the energy reaching the retina is diminished by the absorption in the media of the eye of which we will now take account.

General Nature of Absorption.

By absorption one means in general terms the stoppage of energy in any medium. This may be either specific, affecting only energy of certain wave length, or general, affecting more or less all energy whatever. In the former case it is due to the molecular or atomic structure of the material, in the latter to the fact that it is not physically homogeneous. In specific absorption such as takes place, for instance, in colored glasses, the molecular structure is such as to respond to and take up certain particular oscillation frequencies so that waves of these frequencies do not readily pass through the substance. In general absorption the substance contains particles which reflect the energy from their surface or absorb it without definite regard to its wave length. Such absorption occurs, for instance, in some glass which is full of microscopic bubbles which reflect the energy at their surfaces, or in certain dark glasses which are filled with minute opaque particles. Both kinds of absorption may coexist in the same material, but general absorption involves it only in a very indirect way due to the general properties of reflecting surfaces.

The stoppage of radiant energy in the media of the eye is of two

kinds. First, by absorption in the ordinary sense, and second, by reflection due to the structure of the eye. For instance, the cornea is somewhat lamellar in structure, built up of successive layers and cells and is therefore, owing to the differences in the index of refraction as the ray traverses the structure, somewhat less transparent than if it were homogeneous. There is also a slight loss of energy at the surface in passing into the aqueous. This is also practically homogeneous, but there is again a slight loss by reflection in passage from the aqueous to the lens which has a materially higher index of refraction, and is of itself non homogeneous from the standpoint of refraction. Finally there is reflection passing from the lens into the vitreous and the vitreous itself is not without structure, so that in fact the path of rays through it can be traced by the faint diffused illumination due to its lack of homogeneity. It is quite impossible to determine accurately these losses, except for the initial loss which occurs by reflection at the surface of the cornea to which we have already drawn attention. These losses are greater for rays of short wave length than for those of long, and perhaps the most that can be said about them numerically is that a total loss is probably of the order of magnitude of 10% for rays of medium wave length.

The general absorption of the media of the eye has been studied by Aschkinass [5] in connection with his determination of the absorption spectrum of fluid water. He found that the transmission of the media of the eye for radiant energy in general was closely similar to that of water in a layer of equal thickness. The large proportion of water in these media would, of course, suggest a similarity and Aschkinass found the characteristic absorption bands of water in the experiments on the eyes of cattle and some control experiments on the human eye. The only notable discrepancy was in finding a considerably higher absorption in the cornea than would be warranted by its water equivalent which Aschkinass describes chiefly to a film forming very rapidly over the surface of the dead cornea. In examining the bearing of these facts on the energy focussed upon the retina in any given case it should be noted that the absorption is chiefly in the infra red. Figure 7 shows the absorption curve of a 5^{cm} layer of water as found by Aschkinass [5]. Hence in examining the absorption in any given source of energy it will be found relatively greatest for infra red radiation, except for the effect of the lens in cutting off a large part of the ultra violet. Luckiesh [230] has made a study of the absorption of energy from various sources by the eye, based on Aschkinass's results. From this it appears that from low temperature

sources like carbon incandescent lamps and ordinary flames the absorption of the total energy rises to nearly 90%. As we have already shown this must be increased by miscellaneous losses by reflection so that the amount of energy actually available in the image on the retina from such sources is very small. It is quite otherwise with radiants like the sun, which is roughly equivalent to a body of 5,500 to 6,500 degrees absolute as regards the character of its radiation. From such a source the specific absorption of water cuts off relatively little, and the total loss of energy

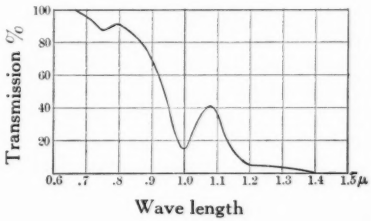


FIGURE 7. Approximate absorption of 5*cm* water, from data of Aschkinass.

in the eye is of the order of magnitude of 25 to 30%. In phenomena like eclipse blindness therefore not only is the eye exposed to a very powerful radiating source, but the radiation is of such character that it is not strongly absorbed and hence the energy in the image may rise to very great intensity. The solar radiation curves already shown make it plain that the proportion of energy cut off will be greater the greater the altitude of the sun and the less the general atmospheric absorption. Taking 30% as the total cut off in the eye one may obtain an approximate idea of the energy concentrated on the retina in observing the sun unscreened, the total radiation being about 10^6 ergs per square cm. per second; and assuming the pupillary diameter to be about 2 mm., approximately 3% of this energy will enter the eye, and subtracting 30% for absorption and reflection it results that the total energy concentrated in the image would be about 20,000 ergs per second. Taking the area affected as approximately .15 mm. in diameter the concentration of energy in the image is on the basis of nearly 113×10^6 ergs per square cm. per second. Even if only a quarter or a half of this amount is available in the case of the partially eclipsed sun, it is evident that the immense concentration of energy in the image is sufficient to produce

destructive effects such as have been often clinically noted and which we have observed in our experiments. From the relatively small absorption by the eye media in the case of solar radiation it is clear, however, that it is far more dangerous, in proportion to its intensity, than any artificial source of radiation.

Passing from the general absorption of the eye for radiant energy here considered to the specific absorption of the several media, the facts have been pretty thoroughly established by the researches of Hallauer[152], Schans and Stockhausen[312] and Martin[238]. As regards the general volume of radiant energy received by the eye there is no specific absorption except that already noted due to the aqueous content. Aside from this the numerical proportion of the energy from most sources specifically absorbed in the eye is very small and is confined to the ultra violet region. The human cornea cuts off practically all the energy of wave length less than 295 $\mu\mu$. The lens wipes out the remaining ultra violet up to a point between 380 $\mu\mu$ and 400 $\mu\mu$. The vitreous absorbs strongly in the general region between 250 $\mu\mu$ and 300 $\mu\mu$ in the thickness in which it exists in the human eye. Only a very minute proportion of energy within this range gets through so that the general effect of the absorption in the vitreous in the case of an aphakic eye is to re-enforce that of the cornea, as is well shown by the immunity from abiotic action of the retina in our experiment No. 89. In his experiment on the eye of a young rabbit, Martin found the limits of transmission to be about those here noted, except that the lens transmitted freely radiations longer than 350 $\mu\mu$. As the human lens yellows with age its absorption reaches down into the violet, extending even to 420 $\mu\mu$.

Eclipse Blindness and Allied Phenomena.

Every recent eclipse of the sun has given rise to numerous cases of so-called eclipse blindness, due to careless observation of the phenomenon in its partial stages, either with the naked eye or with altogether insufficient protection. We should not here consider the matter worthy of attention were it not for the fact that it has been loosely ascribed, like many other imperfectly investigated ocular injuries, to the malign effects of ultra violet light. Eclipse blindness appears in literature as far back as Plato's Phaedo, and is repeatedly mentioned through classical and post classical times as an apparently not unexpected phenomenon. The eclipse of April 17, 1912, in

Germany produced a total of many hundred cases of more or less injury to the eyes, as noted by Wendenberg [410]. Every eye clinic received its toll of more or less severe cases. Clinically the immediate effect is marked and immediate scotoma, which does not pass away promptly but leaves more or less serious cloudiness of vision and accompanying loss of acuity which may be temporary, lasting a few weeks, or in severe cases permanent. The scotoma is commonly central and generally of small extent, in a marked proportion of the cases corresponding fairly well with the dimensions of the sun's image, although wide variations from this may be due to repeated fixations overlapping or reenforcing each other. As it is generally impossible to tell just how long or how often the patient fixed the phenomenon nothing definite can be postulated concerning various varieties of scotoma which have been noted by various observers. The ophthalmoscopic observations usually show changes ranging from scarcely perceptible, to conspicuous and permanent pathological appearances involving lasting and destructive injury to the retina. Metamorphopsia sometimes appears, the significance of which will be apparent in connection with some of our experiments, and diminution of visual acuity is fairly well marked, often falling below one third. With the progress of time the scotoma tends to contract and in mild cases normal vision is regained within some weeks, or in the most severe cases great reduction in acuity persists permanently.*

Our experiments have been directed to the production of an artificial eclipse blindness in animals, and the examination of the lesions produced, following up the work of Czerny [85], Deutschman [89], Herzog [176], and others with special reference to the intensity required to produce the lesions noted. The character of the lesions produced in these experiments is described elsewhere (page 697). The apparatus employed was powerful enough to produce prompt and acute effects. For most of the experiment we employed the mirror apparatus shown in Plate 8 which consisted of a silvered glass mirror 26 cm. in diameter and 1.5 meters focal length, carried, as shown, in a fork mounting set up approximately in the meridian and fitted with slow motions in right ascension and declination so that the beam could be readily

* Jess [200] describes relative ring scotoma for colors in a series of cases, but does not offer a convincing explanation for its occurrence. Boehm [48] was unable to demonstrate it in any of his cases, although he examined them with special reference to it. Birch-Hirschfeld [41] suggests that a normal eye would show the same condition if examined in the same way. This criticism would seem to apply with equal force to the similar scotomata reported by Birch-Hirschfeld [35] himself as occurring after exposure to ultra violet light.

directed and kept in position. In use the mirror was slightly tilted so as to throw the focus just out of the path of the direct incident beam. The concentration of energy obtained by this instrument was enormously great, owing to the size of the mirror and its relatively short focal length. Its area was about 530 square cm. so that with an average reflective coefficient of 0.75, with good sunlight the reflected energy would amount to some 4×10^8 ergs per second. The image of the sun formed by this mirror is 13.2 mm. in diameter or about 1.36 square cm. in area. The energy at the focus then amounts to approximately 30 watts per square cm. A pupil expanded to say 10 mm. by the use of mydriatics would therefore take in a pencil equivalent to about 24×10^7 ergs per second. Allowing as in other cases one-third for the energy absorbed by the media of the eye as a whole, the energy incident in the image would be approximately 16×10^7 ergs per second. The diameter of the image in this case is just over 2.5 mm., corresponding quite exactly to an area of 5 square mm. The energy density in the retinal image therefore would be about 32×10^8 ergs per second per square cm. and it was found in our experiments that an exposure of $\frac{1}{4}$ second to this intensity was sufficient to produce a destructive thermic effect in the retina. This short period is very striking in comparison with the relatively long exposures necessary to produce typical eclipse blindness with the naked eye, although it agrees very well with the data which we later cite regarding energy burns from other sources. The secret of the relative resisting power of the naked eye is that usually in observations of the sun, the pupil is in extreme miosis, so that the amount of energy received is probably not more than 6% of that computed for the normal pupil, while the extremely small area of the solar image favors rapid dissipation of the energy not found when a considerable area is attacked, as in the case of the mirror experiments. The latter condition we have often noted in thermal experiments of other sorts with the big mirror and lenses of various kinds. A concentration of energy very much greater than that from the mirror, acts much more sluggishly on inflammable material when the focus is merely a minute point instead of an appreciable area. Another factor that tends to protect the human eye from the thermic action of light sources of small size, is the impossibility of perfect fixation for any length of time. This is well shown by some experiments on our own eyes (see page 732).

The screens employed in the work are noted in connection with the various experiments. Inasmuch as silver reflects very badly in the region near the extreme ultra violet end of the solar spectrum and it

appeared not desirable to eliminate the possibility of specific injury due to such rays we employed in part of the experimental work as a substitute of the mirror in the concentration of the solar energy a quartz lens 12 cm. in diameter and 25 cm. focal length mounted on an adjustable stand so that we could work directly in its focus. A priori the clinical evidence is strongly against any definite pathological effects due to the ultra violet radiation as such. Numerous cases are recorded in which typical eclipse blindness has been produced through ordinary spectacle lenses, through glass insufficiently dark, and through opera glasses and the like, in all of which cases the abiotic rays are, as we have already shown, cut off. Even very small thicknesses of colored or even clear glasses are sufficient completely to absorb these rays, which moreover are always cut off by the lens so that they cannot reach the retina where the lesions are found. This is in accordance with the conclusions reached by Parsons[265] in analyzing the evidence at hand. Attempts have been made by several investigators, notably Birch-Hirschfeld, to eliminate the infra red rays also by the use of thick water cells and other absorbing media, but these attempts so far as experiments with solar light are concerned are futile, because, as a glance at Figure 5 (page 705) will show, the greater portion of the solar energy lies entirely within the visible spectrum with its intense maximum in the blue or green according to the effect of the atmospheric absorption, so that for solar radiation it is the light rays which are thermally effective, the energy radiation in the ultra violet and infra red being relatively insignificant. Our experiments show with the utmost distinctness that the effects known as eclipse blindness are wholly thermic, due to the intense concentration of the solar energy upon the retina by the refracting system of the eye itself forming an image of destructive energy intensity, the amount of which we have already computed in considering the energy concentrated in images. It is only the briefness of the casual fixations of the sun, and the great reduction of the size of the pupil in response to the intense illumination, that prevents the very common occurrence of such injuries. In the observation of an eclipse the patient is tempted to dangerously long fixation and the necessary results follow.

With long fixation the typical retinal lesions of eclipse blindness may be produced by sources of moderate intensity. For instance in our experiment No. 53 an exposure of twelve minutes was made on the eye of an albino rabbit with the single quartz lens system and the magnetite arc as source, through the 5 cm. quartz water cell. Now from experiments previously made on the radiation from the magnetite

arc by one of us the energy entering the pupil was of the order of magnitude of 444,000 ergs per second. Examination of the retina showed that the lesion produced was practically 3 mm. in diameter. Hence the concentration of the energy in the image allowing the same absorption as in the previous computation amounted to nearly 42×10^6 ergs per second per square cm. This is roughly $\frac{1}{28}$ of the concentration in a direct solar image and correspondingly the lesion produced was comparable with that of a typical case of eclipse blindness. It is quite impossible to get an accurate idea of the critical length of fixation which appears in cases of eclipse blindness, since the observations producing it are generally discontinuous and not noted. This experiment, however, indicates, making due allowance for the extent of the experimental image and for the extremely small size of the pupil in looking at the sun with the naked eye, that the critical period for the development of eclipse blindness is, with close fixation, of the order of magnitude of a minute or less. An exposure of even a few seconds would be highly dangerous were it not for the extreme miosis set up and the usual wandering of the image upon the retina.

Rapid shifting of the focal image on the retina gives the tissue an opportunity for cooling, so that if the fixation at a single point is not long enough to produce destructive effects little permanent damage can be done, although the scotomata may be severe. Our experiments Nos. 100 and 101, in which the exposure to the solar heat through a blue uviol screen was intermittent, show this excellently. In the first the exposure was for alternate seconds over a period aggregating ten minutes, or more than six times as long as necessary to produce burning of the retina in a continuous exposure. No damage was done. The second experiment, in which no water cell was used, consisted of 220 exposures of $\frac{1}{2}$ second with one to three seconds interval between. In this case there was again no damage done although the exposure was three and two-thirds times as long as was required to produce destructive lesions of the retina in two different cases with a continuous exposure.

In this connection one may note that the experiment of Best[27] in fixing the sun for ten seconds through a screen of blue uviol glass was a somewhat hazardous one since this glass lets through a very material proportion of the energy from a high temperature source like the sun. Best's purpose in making this experiment was to show that ultra violet light is not injurious to the retina of a normal eye. The exposure however, was too brief for the result to be of importance in this regard. It is not ultra violet energy which is to be feared in a

case like this as we have already shown, but the danger was from pure heat radiation of which 10% to 15% was due to pass through the uviol glass. We produced in experiment No. 97 most destructive effects from solar heat passing through this medium, and while Best's experiment of ten seconds was below the danger limit it should never be forgotten that the solar energy lies well toward the blue end of the spectrum, and media which successfully cut out the red and infra red are of very little service in protection against solar radiation.

Thermic Effects on the Retina from short Circuits.

It is worth noting in connection with eclipse blindness that sources of intense energy other than the sun may produce similar results. For example, Uhthoff [373] reports the case of a patient exposed to a violent short circuit in which a fortnight after the accident grayish spots due to alterations in the pigment epithelium were observable in the macula of the left eye and were still observable six months later. This ophthalmoscopic appearance is closely similar to that many times recorded in eclipse blindness. Still later Knapp [205] records a case of bilateral injury produced by a tremendously powerful short circuit occurring a scant half meter from the patient's face. There was complete temporary scotoma followed on the next day by some superficial symptoms indicating photophthalmia, and a week later by metamorphopsia, while the vision had been steadily sub-normal. In each fundus was a patch corresponding to the image of the short circuit flare, in which serious damage had been done. These injured areas were still obvious a year later. The retinal lesions described, and especially the metamorphopsia, are such as are typical in the case of eclipse blindness. Here the energy radiation of the short circuit was concentrated in the image to an extent sufficient to produce a typical thermic lesion. The slightness of the abiotic radiation received is evidenced by the very brief superficial symptoms, and the retinal injury, owing to the absorption of practically the whole ultra violet by the media of the eye, must have been due essentially to the pure energy radiation of which the amount, judging by the description, was probably not less than 100 to 200 kw. A short circuit involving 100 kw. would give a superficial intensity at a half meter of over 30,000,000 ergs per square cm., that is, more than thirty times the intensity of solar radiation. The area of the scotoma produced

was, from the description, in the neighborhood of 1 sq. mm. Assuming a pupillary diameter of 5 mm. likely to be found in working in a moderate degree of light when surprised by the short circuit, the energy entering the pupil would be at least 6×10^6 ergs per second concentrated in the image, that is an energy density amounting to in the neighborhood of 6×10^8 ergs per second per square centimeter reckoned without regard for absorption. Allowing one third of the energy absorbed in the eye the energy density in the image should be 4×10^8 ergs per second per square cm. two or three times, at least, greater than the corresponding energy density for a direct observation of the sun, very possibly, owing to the intensity of the short circuit, even several times greater than this. It is little wonder then that although the exposure time is stated to be less than 1 second the results were serious. In true eclipse blindness the length of fixation is the chief factor in the damage.

Thermic Effects on the Retina from Lightning Flashes.

A consideration of these miscellaneous energy effects on the eye would be incomplete without referring to the injuries to the eye received from lightning. In such cases a sharp distinction must be drawn between cases in which the patient is actually struck by lightning, with more or less serious effects, and those in which the patient is clearly not struck, but subject to direct radiation from a nearby flash of lightning. In the former class of injuries electrolytic action and exceedingly severe nervous shock generally occur and the final results may include various grave ocular symptoms sometimes ending in complete blindness due to cataract or atrophy of the optic nerve. In the second class of cases the effects are usually limited to severe scotomata which may impair vision for some hours or days but as a rule there are no lesions visible either superficially or with the ophthalmoscope, and no permanent damage is done. This immunity is chiefly due to the usually considerable distance between the actual lightning bolt and the observer, since the amount of energy actually involved in a lightning discharge of the first order of magnitude may be enormously great. Sir Oliver Lodge estimates it as high as 10^{20} ergs. There are a few instances, however, in which the energy received at the eye has been great enough to produce typical lesions both from abiotic action and probably also from purely thermal

effects. Silex[337], Vossius[407], Rivers[276], Dunbar Roy[302] and Le Roux et Renaud[225] have all noted superficial injuries of the cornea and the last named, as also Oliver[260] have noted symptoms of chorio retinitis, which seem to indicate lesions due to thermic effects, in the latter case invoking the metamorphopsia frequently associated with eclipse blindness. The case of Le Roux et Renaud was a specially notable one in which the patient, on guard duty at night, was exposed to a very powerful flash. It was immediately followed by violent erythropsia which lasted for some two hours. The Gendarme remained at his post and went home and to bed about three hours later. The next morning he woke with acute headache, with substantial blindness in the left eye followed a few hours later by loss of sight in the right eye. There was double acute conjunctivitis with swelling and reddening of the lids and conjunctiva and marked chemosis. A little later there was diffuse interstitial keratitis, a change in the color of the iris from blue to greenish and a grayish haze on the lens front visible in a bright light. These affections of the anterior eye cleared later and when the ophthalmoscope could be used there was marked haze in the vitreous, which cleared very slowly and not completely even after three years. This was believed by Le Roux et Renaud to be associated with chorio retinitis and was certainly secondary to the original lesions.

It is very difficult to make anything like an exact computation of the energy which produced these results, since not only is the total amount of energy in a lightning flash extremely variable and known only to a rough approximation, but the duration of the flash is also variable and uncertain. Thus much is clear, however, that the very heavy discharges, in which the length of flash is some hundreds or thousands of meters and the quantity of electricity discharged very large, are also the relatively slow flashes, since the equivalent condenser capacity is very large. The estimates of frequency rising to millions per second can have no place here, since obviously the velocity of free waves being only 300,000 km. per second, a flash of one or several km. in length cannot have a very high oscillation frequency even supposing it permits oscillations at all. Attempts to measure the frequency of the discharge have often led to results of less than .001 of a second and it is altogether probable that in these long flashes there is no oscillation at all on account of the resistance effects. Starting from the estimate of Sir Oliver Lodge of 10^{20} ergs per second and assuming an effective time of discharge .0001 of a second, and that of the total flash not over .1 is within the effective range of reaching the

eye, the energy in the discharge may be reckoned as one or perhaps several thousand times that of the short circuit discussed in connection with Knapp's case. With a nearby discharge occurring say within 10 or 20 meters, the quantity of energy received by the eye would be amply great to account for even severer results than those noted by Knapp (loc. cit.).

Working back from our experiments with the bare magnetite lamp carrying about 500 watts in the arc, it again appears that the quantity of energy assumed by Sir Oliver Lodge is more than sufficient to account for the results of a nearby discharge. It therefore must be admitted that the direct action of lightning in producing both abiotic and thermic lesions in the eye as in the cases of Le Roux et Renaud and of Oliver is well within the bounds of possibility although requiring very unusual proximity to a powerful discharge which may well have been the case in these instances. The extreme rarity of such clinical conditions is perhaps ascribable to the few instances in which there is close proximity to the stroke combined with free exposure of the eyes without the patient being actually involved in the shock. One must consider therefore that lesions produced directly by the radiating energy of a lightning discharge are extremely unusual and unlikely to occur, although well within the range of possibility, as the cases here referred to show.

Possible Specific Action of Infra Red Radiation.

As we have already stated, there was no segregation of various radiations in our experiments on thermic effects except in so far as abiotic radiations were cut off by certain screens. So far as all indications go the effect of all other radiation than the abiotic is chiefly chargeable to thermic energy without respect to wave length. As already explained different sources present totally different distributions of energy with respect to wave length, the lower the temperature the greater being the proportion of the so-called infra-red rays. In the case of the quartz mercury arc and the magnetite arc with which we chiefly worked, the spectra are essentially discontinuous and hence do not obey Planck's law, so that there is no definite relation between the temperature and the wave length of maximum radiation.

The total energy spectrum of each of these sources, however, is exceedingly complex. Of the total energy spent in the arc a certain proportion goes to maintain the characteristic linear spectra, a cer-

tain other portion goes to heating the electrode or containing tube and the surrounding mechanism. While therefore the spectrum which can be seen or photographed is linear, there is superimposed upon it the continuous spectrum of the radiating solid at rather low temperature and of relatively large extent, since the radiation is not only from the arc or its containing tube but from the immediately heated surroundings. Much of the loss of efficiency in both sources referred to comes from this secondary heat radiation. This is particularly the case in the quartz mercury arc of which the actual light-giving efficiency is very high, much higher than is indicated even by its really small specific consumption per c. p.

The existence of this secondary radiation which is mainly of very long wave length, make comparisons between such sources and the ordinary radiating solids very difficult. The following table gives for the magnetite arc the transmission with respect to the total energy of the most important of the various media which we employed, as determined by a Rubens thermopile.

ABSORPTION OF CERTAIN SCREENS.

Source Magnetite Arc.

Filter			Percentage of Transmission
2 quartz plates each 3 mm. thick			53
Same + 5 cm. distilled water			33
Water cell and Dense Flint N_D	1.69	(335 $\mu\mu$)	26
" " " Medium Flint N_D	1.62	(315 $\mu\mu$)	28
" " " Light Flint N_D	1.57	(305 $\mu\mu$)	27
" " " Crown N_D	1.51	(295 $\mu\mu$)	28
Dense Flint N_D	1.69	alone	40
Medium Flint N_D	1.63	"	45
Light Flint N_D	1.57	"	40
Crown N_D	1.51	"	43

It will be observed that the actual transmission of the empty quartz cell consisting of two 3 mm. polished plates was only 53% of the total energy. Of the 47% lost, roughly 15% should be in the reflections from the four surfaces of the two 3 mm. polished plates. The remainder, that is more than a third of the total energy, is mainly

from the cut-off of secondary radiation of relatively very long wave length, 4 to 5 μ and more, received from radiating surfaces at and below red heat. The addition of 5 cm. of distilled water in filling the cell reduced the transmitted radiation to 33%, which represents the radiation between the former limit and approximately 1 μ. There is every reason to believe that most of this energy is from the hot body radiation rather than from the line spectrum of the arc itself, for in so far as known metallic spectra are not rich in intense infra-red lines. Screen No. 1 then, cuts off 22% of the energy between 1 μ and the extreme ultra violet. Screen No. 2, medium optical flint, is relatively transparent, almost as transparent as the light crown screen No. 7, while the light flint screen No. 4 cuts off more energy than either of these. Used without the water cell all the four screens mentioned cut off between 50 and 60 % of the total energy due to the large absorption of glass in the extreme infra red corresponding to the secondary hot-body radiation of the source.

This secondary radiation being from a very diffused source cannot have a conspicuous effect in those experiments in which the light was concentrated through lenses although it comes into play in the free radiation from the arc. It is quite certain, for instance, that in the bactericidal experiments with the mercury arc which one of us has recorded, in which trouble was experienced from heating of the water in which the bacteria were suspended, this heating was mainly from the large secondary radiation which is readily stopped by water rather than from the characteristic radiation of the mercury vapor.

The data heretofore given by one of us on the proportion of ultra violet energy in the quartz mercury lamp and the magnetite arc may be regarded as substantially correct for the metallic spectra as such, the quartz water cell of 1 cm. thickness employed in these experiments cutting off the secondary radiations rather completely without interfering materially with the energy of the line spectrum. In our experiments involving total thermic effects necessary corrections for the conditions of the experiment can be made by reference to the foregoing table.

As regards the thermic action of radiation on the eye, there is no reason to suspect any specific effects with respect to wave length. So far as the action is not definitely abiotic or concerned with the stimulation of the light perceiving functions of the retina it seems to be purely a question of energy as in any other case of heating. The more violent phenomena produced by heating are considered in our discussion of eclipse blindness and allied phenomena (page 720).

The possible effects on the lens of heat radiation persisting over a very long period we have considered in discussing glass blowers cataract (page 734). Thermic effects on the cornea we have shown to be obtainable only under extreme experimental conditions (page 692). The media of the eyes generally, as we have already shown, are of substantially the same absorbing character as water so far as concerns visible and infra red rays and therefore take up chiefly radiant energy of long wave length such as is prominent in radiation received from low temperature sources like hot metals or molten glass. The pigmented iris and the pigment epithelium of the fundus are exceptions in that they absorb quite completely radiant energy irrespective of wave length. There is, therefore, a tendency to produce localized thermic effects in both these structures, as we have shown in various of our experiments. The lesions occurring under these circumstances have already been described. In case of the iris, the heat effects may proceed all the way from moderate irritation to serious permanent injury (see page 696). This phase of the matter was recently investigated by Reichen[293]. This observer studied the effects of concentration of infra red radiation on the eye by cutting off the visible and ultra violet spectrum by a filter of iodine in bisulphide of carbon, and absorbing the extreme infra red by a water filter. In this way the rays with which he was concerned were substantially those between 800 and 1200 $\mu\mu$. Using as source the electric arc between carbon electrodes, by means of a rock salt lens he concentrated the filtered energy upon the eyes of rabbits for periods from 1 to 33 minutes. The only effect noted was contraction of the pupil lasting some hours and generally slight, evidently depending on the direct heat effect through the filters used, since the visible rays were practically excluded from the retina. Such pupillary contraction is reasonably to be expected after moderate irritation of the iris, such as would be furnished by this heat stimulus and may occur as some of our experiments show, in the absence of any recognizable histological changes. Violent and persistent effects were hardly to be expected inasmuch as the source used is not rich in rays transmissible through Reichen's filters. Most of the energy from a carbon arc is intercepted by a water filter and a good deal of the remaining energy by the iodine in carbon bisulphides. This selective action is well shown in one of Reichen's experiments in which the water screen, which was quite close to the arc, was boiling violently after $7\frac{1}{2}$ minutes exposure. Reichen's experiments, therefore, merely show a mild irritation not in the least peculiar to the region of the spectrum employed. It would seem quite im-

possible to obtain even this effect except through strong focussing of the light upon the eye, a condition which is not found in the use of ordinary illuminants for any purpose. So far then as infra red radiation is concerned the eye is not subject to any special dangers, and the concentration of heat upon it in any way would inevitably set up a danger signal of painful sensation long before any definite heat effects could be obtained.

In fact it is easily demonstrable that the full radiation that can under practical conditions be received from the most powerful illuminants is incapable of producing on the retina any lesions due to thermic action such as may be found in eclipse blindness. In our Experiment 53, already referred to, changes in the pigment epithelium and a definite burnt area were produced. The diameter of the area in which histological changes were clear was 3 mm. shading off toward the edges and having a central area about 1 mm. in diameter, in which the damage was serious and comparable with that in eclipse blindness. We have found that in this case the concentration of the energy in the image amounted to very nearly 4.2×10^6 ergs per second per square cm., roughly $\frac{1}{26}$ of that found in the solar image formed directly through a 3 mm. pupil. The exposure to this intensity lasted 12 minutes. Here then is a definite case in which the result was positive. Negative results, however, of which a few were obtained in our experiments, are unsatisfactory since they do not take account of the wandering of the image, or of imperfect fixation which as we have shown would be likely to avert injury as in the typical case of intermitted exposure to heat.

Our large magnetite arc gives at a distance of 2 meters a total radiation of about 2500 ergs per second per square cm. At this distance from the arc we find the pupil of the human eye is narrowed to a scant 2 mm. and this area would intercept about 75 ergs per second of the total amount stated, allowing $\frac{1}{3}$ absorption as heretofore. This means that the energy concentrated on the retina would be about 50 ergs per second. To compute the energy density requires a knowledge of the size of the image including the element of imperfect fixation. We have found from personal experiments that on fixing at 2 meters the magnetite arc for a few seconds and measuring the size of the scotoma produced by viewing at the same distance a card ruled to centimeters, that even for this short fixation period the area of the scotoma is nearly four times that of the geometrical image of the source. On fixation of 6 minutes the scotoma rises to twenty-five times the size of the geometrical image. This latter has an area of

about 0.01 square mm. as determined from photographic data, so that for a long fixation the energy received would be distributed over practically $\frac{1}{4}$ square mm. of area. The density in the image as actually obtained in a long fixation would amount to about 20,000 ergs per second per square cm., only about $\frac{1}{200}$ part of the energy density which produced the positive result in experiment No. 53. This difference is still further enhanced practically by considerable difference in the size of the image areas, the smaller one being relatively less effective than the large on account of the more rapid dissipation of heat. It is therefore evident that an exposure of even 12 minutes with close fixation to a source as powerful as this arc, a thing which no rational human being would be likely to undertake, still involves only a small fraction of the minimum energy known to produce definite lesions.

Further evidence of the harmlessness of such exposures even to very powerful artificial illuminants may be derived from noting that the heat concentration in this case, that is 20,000 ergs per second per square cm. is less than $\frac{1}{5000}$, of that received by direct fixation of the sun with the same pupillary aperture (see page 719). Now it is well known that one can fix the sun for a very few seconds without danger of anything more than a temporary scotoma. From this we may conclude that the arc here considered may be fixed, as well as it is possible to secure fixation, for a considerable period without running danger of a permanent scotoma.

In order to demonstrate beyond any possibility of doubt that the retina cannot be injured by exposure of the eye to a source such as the magnetite arc, one of us has actually fixed his eye upon this arc at a distance of two meters for 6 minutes on two occasions. The macula itself was not fixed upon the arc but upon an object of the same size placed 2.6°, to one side of it. In this way more perfect fixation of the image of the arc on the retina was ensured than if the arc had been looked at directly. The results were only of a temporary character. There was considerable xanthopsia lasting about 3 minutes. The scotoma had disappeared after each observation in less than ten minutes and could only be detected for a few hours by careful dark adaptation of the eye. The following day no traces of it were determined. It is therefore clear from actual experiment as well as from general principles that even the extremely powerful arc which we have here used is from the standpoint of injury to the retina entirely harmless under any circumstances conceivable in practical use. Some large carbon arcs may yield even two or six times the total heat energy

of the magnetite arc here in question while yet remaining within perfectly safe limits from any practical standpoint. No actual artificial illuminant can fairly be considered dangerous from the standpoint of thermic action on the retina. It will be observed that since no screens were used, these experiments also afford evidence of the harmlessness of abiotic radiations to the retina.

Glassblowers' Cataract.

The fact that glassblowers are subject to a special form of cataract has naturally raised the question whether or not the latter is due to radiant energy and if so, whether to abiotic or thermic action. Meyhofer [245] examined 506 glassmakers and found 11.6% affected with cataract. The cataract almost always appears first in the left eye which is the more exposed to the light. When it appears first in the right eye it has been found that the glassblower has been in the habit of turning this eye towards the oven (Stein [349]). The glassblowers show also a peculiar rusty brown spot on each cheek, more marked on the left. The length of time necessary for the development of the cataract has not been exactly ascertained, but it is evidently several years. The cataract usually begins before the age of 40. In Meyhofer's series the youngest blower was aged 15 years and the youngest affected with cataract 17 years. Of 59 cases of cataract, 42 were under the age of 40. In the latter cases, both eyes were affected in 16, the left eye alone in 19, and the right eye alone in 7. The glassblowers are thin and delicate, and are subject to asthma and pulmonary tuberculosis. Almost all of them have emphysema of the parotid gland. During their working hours they perspire excessively and in consequence drink enormous quantities of fluids, including beer, coffee, wine, and lemonade.

The cataract begins as a rosette like or diffuse opacity in the cortex at the posterior pole of the lens, the remainder of the lens for a long time remaining clear. Later, striae similar to those of senile cataract may appear. At operation the nucleus is found larger than that of other individuals of the same age (Stein [349]), and the capsule more fragile (Cramer [81]). Parsons [265] states that in only one-fifth of the cases does the cataract differ in appearance from a senile cataract, but this probably applies to the late stages.

Hirschberg [192] states that for over 100 years it has been recognized by various observers that individuals exposed to intense heat and light

are especially liable to cataract. Peters[272] suggested that the cataract was due to the venous stasis in the vortex veins associated with the forced expiration, analogous to the cataract produced experimentally by tying off the vortex veins. Leber[222] advanced the view that it was due to concentration of the aqueous resulting from evaporation from the cornea and the loss of water from excessive perspiration. Parsons[265] suggested that the cataract results from altered nutrition due to overheating of the ciliary body. Cramer[81], Stein[349], and others believe that it is due to ultra violet light (chemical action), while Vogt[399] regards the infra red rays as chiefly responsible.

The great frequency with which glassblowers' cataract occurs, its relatively uniform type, and the fact above all, that it occurs first in the more exposed eye, show clearly enough that it is chiefly due to the action of radiant energy on the eye itself. This is supported also by the fact that the cheek shows a more marked area of discoloration on the side of the first affected eye. The further questions whether the cataract is due to the direct action of the light upon the lens, or upon the eye as a whole, and whether it is due to abiotic or thermic action, are not quite so easily answered.

The character of the radiation from molten glass is well known. It is that from a homogeneous body of relatively low temperature, 1200° to 1400° C. It is certain that the spectrum of a non gaseous body at this temperature does not include any of the so-called abiotic radiation since the extreme limit of the spectrum of molten glass found by any investigator is 320 $\mu\mu$ and estimates range from that to 334 $\mu\mu$. We have already shown that the abiotic action cannot be traced beyond 305 $\mu\mu$ and there is not the slightest indication from our researches or any predecessors that there is reason to suspect an extension of such activity to waves longer than 320 $\mu\mu$. Even if there were, such rays would be stopped at the front of the lens by its absorption and hence would be unable to affect the posterior cortex. Moreover the radiation of a body at such temperature is relatively very weak all through the ultra violet, the maximum, according to Planck's law, for a body at 1300° C. lying far in the infra red while the energy in the whole visible and ultra violet part of the spectrum is less than 1% of the total. Hence to ascribe injurious effects to the visible or ultra violet radiation without eliminating once and for all the 99% of infra red radiation is to lose all sense of proportion between cause and effect. To follow up the theory of the matter a little further we have shown that the specific abiotic action is clearly to be eliminated. Of the rays which are

absorbed by the lens, reaching from 300 $\mu\mu$ to about 400 $\mu\mu$, the chief absorption is, following the general theory which we have already explained, at the front surface, hence if by any stretch of the imagination glassblowers' cataract could be assumed to be due to an indefinitely long application of such rays it should occur at the anterior and not at the posterior cortex. To rays in the ordinary visible spectrum the lens is notoriously transparent and in default of absorption of energy there is no reason to expect any specific effects from it. We have been able by the use of sources of extreme power greatly concentrated, as our experiments show, to obtain specific action of the ultra violet rays only to a microscopic depth, 20 μ, so that the experimental evidence lies squarely against any lesions directly producible by such sources in the posterior cortex, particularly in the absence of any effects in the anterior cortex.

This reasoning also holds for the time integrals of any effect of such rays over periods however long, for whatever the aggregate effect might be it would always remain much greater at the anterior than at the posterior part of the lens substance. The absorption of the media of the eye for various wave lengths and especially those predominant in sources of the temperature considered has been investigated by Aschkinass [5] who has shown that the characteristic absorption of the long waves is essentially that of water. An analysis of Aschkinass' results by Luckiesh [230] shows that for sources of moderate temperature the resulting absorption is chiefly in the cornea and aqueous, that is in the outer layers of the absorbing media. For a source of the temperature of a glass furnace, between 80 and 90% of the energy will be absorbed by the cornea alone, and the amount stopped in the lens from all causes would not be over 3 or 4%, but this small fraction of the total is still absolutely a considerable quantity and is somewhat concentrated in the lens since not less than three-fourths of the total refraction of the eye is due to the cornea. There is therefore a slight tendency to concentrate energy toward the rear of the lens. Such concentration must be small, however, owing to the contracted pupil and the resulting narrow angle of the pencil of rays entering the lens. It is doubtful whether the actual concentration would reach more than a few per cent and the effect of this would be more than offset by the greater absorption in the anterior cortex.

As regards the distribution of temperature in the eye resulting from the intense radiation most of the absorption takes place as stated, in the cornea, and a greater proportion in the aqueous than in the lens. The front of the cornea is of course rapidly cooled by convection and

the fluid of the anterior chamber also gets some facility at cooling by convection currents. The iris which strongly absorbs most of the energy which falls upon it especially if strongly colored, to a certain extent screens the front surface of the lens behind it especially since the circulatory system in the iris tends to prevent its temperature rising materially unless the access of energy is above the rate at which circulation can take care of it. At the rear of the lens the vitreous with its fibrillated structure effectively prevents, like all such substances, the existence of convection currents. Just what the net effect of the structure is upon the steady distribution of temperature when the eye is exposed to radiation cannot be quantitatively determined, and while undoubtedly the heat reaching the rear of the lens from the energy transmitted to that point, or received from that taken up by absorption in the anterior part of the eye, cannot readily escape and hence tends toward concentration it seems somewhat doubtful whether this cause alone could determine the starting of cataract at the posterior cortex. We are inclined to attach more importance to the suggestions of Leber [222] that the effect is a secondary one due to the loss of water in the drain produced by the heat on the front of the eye and elsewhere, and especially to that of Parsons [265] that malnutrition due to interference with the functions of the ciliary body by the heat may be chargeably with the malady. We are the more inclined to this opinion since the intense radiation acts through the sclera as well as through the cornea, thus affecting the whole structure. It is also a well known clinical fact that diseases of the fundus produce at times cataractous changes in the posterior cortex that are held to be due to impaired nutrition of the lens. The development of glass blowers' cataract is so slow that it is quite hopeless to reach its cause experimentally, but from the facts here stated we incline to the opinion that these secondary effects of radiation are more important in producing it than the specific action of radiation in producing localized effect at the posterior cortex. In any case it is perfectly clear that abiotic radiations are not concerned, and have nothing to do with the matter.

Applications to Commercial Illuminants.

In considering any possible deleterious effects of radiation upon the eye there are certain pathological effects which can be at once eliminated, at least from any consideration of commercial illuminants.

First, by the experiments which have been heretofore described we have made it clear that there can be no injury done to the retina by ultra violet light as such, even the most severe exposures failing completely to produce any effect whatever. Second, all thermic effects of energy from any source on the external eye are at once ruled out of consideration by the immediate discomfort produced by excessive heat. No person would tolerate extreme heat radiation on the external eye for a period long enough to produce the slightest damage. There remain therefore to be considered thermic effects within the eye, and specially those due to the focussing of intense radiation upon the retina as in eclipse blindness; over stimulation of the physiological processes in the retina, that is pathological effects due to light as such in its action on the retinal structure, and finally those abiotic effects of the extreme ultra violet rays on the external eye properly known as photophthalmia. It has been our purpose to ascertain the practical risks incurred in the use of artificial illuminants and the precautions required to avoid danger. To this end have we sought especially the quantitative relations in the action of radiant energy upon the eye. We have, therefore, experimented with the most powerful sources used for practical lighting under conditions of intensity immensely greater than occur in their every day use. We have shown (page 728) that as regards the general thermic effects of energy upon the eye there is no chance of damage to the retina or to the media of the eye under any practical conditions of use. With respect to damage to the retina in particular we have been unable to produce it except by exposures and intensities enormously greater than could possibly be reached in the use of artificial sources of light. We have used beside the quartz mercury arc which is not particularly strong in general radiation, a 750 watt magnetite arc which takes the greatest amount of energy of any arc light ordinarily used for illuminating purposes and the 750 watt nitrogen lamp which gives more ultra violet than any other incandescent source, and which failed completely to produce any specific damage to the eye, although in one experiment the animal was overcome by the general heat effect, as in sunstroke. This occurred after an exposure of $1\frac{1}{2}$ hours at 20 cm. from the filament. A second experiment with an exposure of 2 hours at the same distance, in which the animal was well protected from the heat and the head kept cooled with water, showed no damage to the eye of any kind. This source as a whole focusses less sharply than does the arc lamp taking an equal amount of energy, and in this case we have already shown that the retina would not be subject to damage

save by an impossibly long exposure with accurate fixation. These experiments were at far shorter distances and consequently of enormously greater intensities than any which could be found with such sources as illuminants, and we may hence conclude that so far as general effects of thermic energy are concerned no source used for illuminating purposes is capable under working conditions of producing any observable deleterious results.

Experiments with the arc lamp are crucial because this source is nearer to being a point source than any illuminant of similar power and hence gives much sharper concentration of energy in the image. It is with this concentration in the image that the possible damage to the retina is concerned. Diffuse sources which do not come to a definite focus may be entirely neglected for this particular purpose. Low temperature sources of which most of the energy lies in the extreme infra red are even less effective in concentrating energy upon the retina, because the eye never focusses such wave lengths on the retina except as a diffuse spot.

The only sources from which there seems to be any material danger from the standpoint of thermic effects are certain very powerful high temperature sources used in the arts, such as heavy arcs used for welding purposes, furnaces, electric or other, where there is customarily great concentration of energy, and such purely accidental phenomena as short circuits. Perhaps some of the very powerful arcs used in searchlights might be included in this class of the possibly dangerous. Ordinary discretion in avoiding disagreeably powerful lights or suitably shading the eyes from them should avert easily all real danger from any of these sources of radiation, save the short circuits which are accidental rather than ordinary risks. Certainly no sources used for lighting purposes can be classified as dangerous from the standpoint of thermic effects.

We wish particularly to emphasize the fact that so far as any possible temporary or permanent injury to the retina is concerned such action must depend on the concentration of energy in the image. Consequently, extended sources of moderate intrinsic brilliancy are to be preferred to intense sources. Hence it is desirable to protect all sources naturally of high intrinsic brilliancy by diffusing globes.

As regards dangers of injury to the eye from light radiation as such, our experiments indicate that it has been very greatly exaggerated as regards its pathological possibilities. It is undoubtedly true that brilliant sources of light are disagreeable and that they produce unpleasant effects in temporary scotomata, disturbance of color

vision, persistent and annoying after images and fatigue due to efforts to overcome the difficulties of vision under these disadvantages. As regards definite pathological effects or permanent impairment of vision from exposure to the luminous rays alone we have been unable to find either clinically or experimentally anything of a positive nature. The experiments on monkeys, which we have recorded, show very clearly that exposure to light of intensity many times greater than anything to be found in the use of commercial illuminants produced only temporary scotomata. The lid reflexes appeared within a very few minutes and the scotomata seemed to have worn away within at most a few hours. There was not the slightest sign of permanent impairment of vision. As noted on page 684 there were indications that the process of light adaptation may go on to a considerable degree even during very severe exposure to light. In the experiments on the human eye results were found closely comparable with those obtained in the earlier experiments on monkeys. The erythropsia passed away in a few minutes and the scotoma wore away rapidly so that after three hours the visual acuity was normal, although there still remained traces of color scotoma. After 22 hours visual acuity remained normal and the central color vision for red, blue and green was also normal. This intensity of the light in this case was far in excess of anything which could be reached in the use of commercial illuminants and there was a length of fixation many times greater than could ever be found in practical use of lights. These experiments seemed conclusive in showing that the effect of even extraordinarily severe exposure to luminous rays produces only such temporary effects as might reasonably be expected and is followed by no lasting injuries of any kind. Whether frequent and long exposures of a similar kind might exhaust the extraordinary recuperative powers of the eye is a matter on which in the nature of things there can be no direct experimental evidence and which is not of practical importance since under no working conditions could even a single exposure comparable in severity with those obtained in our experiments be produced. There is, however, very strong clinical evidence that even severe daily exposures to intense light lasting over many years fails to produce material injury to the eye. For in the case of glass blowers there is extreme exposure both to the heat and light of the furnace occurring daily for many years. While glass blowers' cataract, probably arising as we have shown from secondary causes quite aside from the direct effects of radiation, may be produced under these circumstances, there has never been noted any injury to the retina. The fact that

the retina is uninjured under these extreme conditions seems to indicate as do our experiments that the eye is remarkably tolerant of intense light even under the circumstances of exposures of great severity lasting over very long periods of time.

These results do not justify the use of powerful unscreened sources near the eye, since in this condition vision becomes difficult and the effects of eye-strain due to other causes than mere illumination of the retina become unpleasantly in evidence. They do show, however, that the eye in the process of its evolution has acquired the ability to take care of itself under extreme conditions of illumination to a degree hitherto deemed highly improbable, and that the effects on the retina due to any exposure to intense light in the least degree likely to be found in the use of practical illuminants are temporary and of no pathological significance. *A fortiori*, there is not even a remote chance of pathological effects on the structure of the eye due to light received from extended surfaces of low intensity like translucent globes and diffuse reflections as from paper.

As for the ultra violet part of the spectrum to which exaggerated importance has been attached by many recent writers, the situation is much the same as with respect to the rest of the spectrum, that is, while under conceivable or realizable conditions of over exposure injury may be done to the external eye yet under all practical conditions found in actual use of artificial sources of light for illumination the ultra violet part of the spectrum may be left out as a possible source of injury. All illuminants possess an easily measurable amount of ultra violet radiation ranging, as one of us has already shown [19] from about 4 ergs per second per square cm. per foot candle of illumination in the quartz arc with the usual globe, to more than twenty times this amount in the enclosed carbon arc shining through a quartz window. Between these two lie the whole range of incandescent lamps both gas and electric, the ordinary mercury arcs and the ordinary Cooper Hewitt tube, flames, arc lamps of various sorts, and sunlight. The last mentioned occupies an intermediate position between the high efficiency electric incandescent lamps and the older incandescent lamps or ordinary flames. The ultra violet in these various sources is distributed in different ways. All the flames and incandescents give continuous spectra which die out for even the highest temperature of these sources at about wave length 300 $\mu\mu$. Sources giving discontinuous spectra generally extend below this limit of wave length, but often with very feeble radiation in this region. Such, for instance, is the case with the carbon arcs, which show chiefly metallic

impurity lines within the very short wave lengths and owe their considerable proportion of ultra violet to radiation just outside the visible spectrum. From the standpoint of effects upon the eye the ultra violet region may be divided into two sharply separated portions, one of which produces abiotic effects while the other does not. We have for the first time definitely established the line of partition between these two portions at 305 $\mu\mu$. Some of the earlier experimenters in this field imagined that they had detected abiotic effects with slightly longer wave lengths, an error apparently due to insufficient knowledge of the absorbing screens which they employed, which with rare exceptions are described, if at all, in very loose terms.

No injurious effects have been attached with any reasonable degree of certainty to the ultra violet radiation which lies between the end of the visible spectrum and the beginning of the abiotic rays. Since this range of radiation is present in considerable amount in ordinary sunlight, it is sufficiently obvious that any definitely harmful results producible under ordinary conditions would have been eliminated by the ordinary progress of evolution. Artificial illuminants under any practical conditions of use expose the eye to much less severe radiation in this part of the spectrum than does ordinary daylight and *a fortiori* can be excluded as possible sources of harm.

With respect to abiotic radiations we have every reason to acquit on sound experimental basis every known artificial illuminant when working under the ordinary conditions of commercial use. Even the quartz mercury lamp, which is per se richer in abiotic radiations than any other commercial source of illumination, when equipped with its ordinary globe is not only less rich in ultra violet per candle power given than any other source, but is as we have found by experiment incapable of producing any abiotic effects on the eye even after six hours exposure at 30 cm. from the tube. We have further shown that even where the lamp is used without its globe, a condition which is avoided in consideration of efficiency, long exposures would still be necessary to produce any injurious effects at any distance reasonably to be expected. The same immunity from danger attaches to all the sources at present in commercial use. It is well within the bounds to say that there is no commercial illuminant from which the least risk of abiotic radiation is incurred under the circumstances of practical use. The only sources used in the arts which have abiotic power enough to require special caution in their use are those not employed for the purpose of illumination. Such, for example, are the powerful arcs used in some electric welding processes, those employed in the

fixation of nitrogen from the air and lamps specifically designed for abiotic purposes, like the ultra violet lamp of Henri, Helbronner and de Recklinghausen.[164] The last mentioned is a quartz arc of peculiar form taking 1150 watts and giving approximately six times as much abiotic radiation as the lamp used in our experiments. Such a source would therefore give typical photophthalmia in about one minute at 50 cm. and milder symptoms of conjunctivitis and erythema in somewhat less than this time. A comparable degree of activity is indicated for the other sources here referred to. In these cases considerable caution must be exercised to avoid even short exposures to the unscreened source, but arcs of this character are highly special in their functions and have no connection with matters of illumination.

As regards the general effects of the ultra violet portion of the spectrum, it must be remembered that the abiotic action is chiefly superficial and we have shown that even under exposures of great severity there are no indications of any injury to the retina from ultra violet rays even in the aphakic eye.

In order to make it clear that the results as to abiotic action, which we have obtained from animal experimentation, are substantially applicable to the human eye as well, we may from the standpoint of general theory point out that the effect of the abiotic rays we have shown to be definitely dependent on the quantity of the radiation, the action of which can be reckoned much as if it were a mere mechanical force. There is no reason from our experiments or those of others to suppose that such radiations act with much greater intensity on one kind of living cell than on another. We have in addition ample direct evidence that the effect on the human eye and on the rabbit's eye are entirely comparable, for we have shown experimentally that the critical amount of abiotic radiation for photophthalmia on the rabbit's cornea and for erythema on the human skin is the same. As a clinical fact, of which the early observations of Charcot[15] are typical, in every case of photophthalmia the erythema of the skin surrounding the eye is quite as conspicuous as the conjunctivitis. In fact, while in practically every case of photophthalmia erythema appears, it is very rare in clinical cases to find the stippling of the cornea taken as one of our characteristic symptoms, hence the amount of abiotic energy required to produce photophthalmia, since it will also produce erythema, must be substantially as great for the human eye as for the rabbit's eyes on which most of our experiments were performed.

Our general conclusion, therefore, regarding the effect of radiation

from practical illuminants on the human eye is that no sources commercially employed for such a purpose are to be regarded as dangerous and that the most ordinary care in providing illumination with which comfortable vision can be obtained is sufficient for complete security against all possibility of injury from radiation.

Protective Glasses.

As we have shown, the lens completely screens the retina from abiotic radiations so that attempts further to protect it from such radiations by means of glasses of any kind are superfluous. We have also shown that the retina even of the aphakic eye, under ordinary conditions is in no danger of injury by any source of light in common use, and is no doubt completely protected by the thick cataract glasses usually worn. In addition we have shown that the retina under ordinary conditions is in no danger of injury from the heat generated within it by the light from such sources. Heat effects are to be feared only in the case of extreme light intensities such as direct sunlight, and, exceptionally, short circuit arcs and lightning flashes. Against these any of the extremely dark glasses are effective.

As regards the external eye, as just pointed out, it also is in no danger from abiotic radiations from any of the usual light sources. Photophthalmia may of course readily be produced by sufficiently long exposures at close range to high power arc lights of any kind, or to the quartz or uviol mercury vapor lamps, but it is only under special conditions that such exposures would occur. Here ordinary spectacles of crown glass usually afford sufficient protection, and adequate protection would certainly be afforded by any of the ordinary yellowish, greenish or grayish protective glasses in common use, preferably in the form of coquilles so as to exclude lateral light. These would also afford ample protection against snow blindness or the photophthalmia produced by short circuits. Birch-Hirschfeld [35] states that from his own personal experience in protracted and regular working with the uviol lamp he found complete protection from photophthalmia with the smoke gray spectacles, and intimates that Stockhausen's claim of having photophthalmia after working with the electric arc lamp, in spite of the fact that he wore ordinary spectacles, is readily explained by the circumstances that common spectacle lenses do not adequately protect the eye from radiation entering laterally.

For protection of the external eye against extreme heat such as

that to which glass blowers are exposed, few glasses except such special ones as have recently been devised by Crookes, are very effective, the deep green copper oxide glasses being perhaps as good as any. Such glasses combined with amber or yellow green tints so as to reduce the transmitted light to a moderate amount in the middle of the spectrum are probably the most efficient protection against extreme radiation of all kinds, except that of direct sunlight. In the case of glass blowers, it is difficult and perhaps futile for them to make use of any sort of glasses, owing to excessive perspiration.

So far then as concerns actual injury by light, the eye under ordinary conditions of modern life is in no danger. The question of wearing protective glasses so far as concerns the ordinary individual therefore narrows itself down to the determination of those best adapted to obviate the sensations resulting from too intense illumination. These are unpleasant in the same way as are extremely loud noises, and for protection against them one wears glasses that reduce the light as one plugs the ears with cotton in a boiler factory. Any glass that reduces the light is effective for this purpose, but preferably, perhaps, a glass that transmits light chiefly in the middle of the spectrum, for which the eye is customarily focussed. The question of the color of the glass is, however, of little importance and the personal idiosyncrasies of the individual may be safely allowed free play here. It is probable that if such glasses are too long worn they will increase the sensitiveness of the individual to light.

The question of the use of protective glasses in pathological conditions of the eye does not specially concern us here, but it may be stated that it is one simply of reducing the intensity of light reaching the retina. In cases of iritis this is possibly of some importance since it favors dilatation of pupil. In cases of glaucoma, on the other hand, excess of light is desirable, since it contracts the pupil. As Hess [184] has pointed out, the sensations incident to the so called photophobia, associated with keratitis and other irritable conditions of the eye, are present in the dark as well as in the light, so that it is evident that undue importance has been attached to the exclusion of light in these conditions. As regards fundus conditions, the use of protective glasses has no rational basis, except possibly in the case of retinitis pigmentosa and allied conditions, as suggested by Axenfeld [8].

The exaggerated attention that has been paid in recent years to the harmful effects of ultra violet radiations has had one good effect in modifying the character of the protective glasses prescribed by ophthalmologists. Earlier practice was based on a general desire to

cut down the light received by patients whose eyes were for one reason or another sensitive. This requirement resulted in the production of glasses of more or less dark neutral tints and sometimes dark shades of colored glasses commercially obtainable like green and blue. A later phase of practice reflected the view common a quarter century ago that red light was a thing producing in some unknown way specially bad effects and consequently was to be shunned. Hence it was not uncommon to prescribe glasses of cobalt blue, green, and various amethyst tints. It is interesting to note that such glasses, while they reduce greatly the red of the visible spectrum still transmit quite freely the nearer part of the infra red which carries a large amount of energy (Coblentz [76]). In fact, of this nearer infra red such glasses transmit almost as much as does ruby glass. Therefore, the earlier protective glasses were not effective in cutting off heat radiation and tended to transmit mainly light toward the blue end of the spectrum. Perhaps the chief benefit of the agitation that has taken place within the last decade on the possible, though as we have shown highly improbable dangers of the ultra violet, has been the bringing into prominence the new types of protective glasses.

These, intended primarily for eliminating the ultra violet rays, have tended to types of selective absorption which give advantageous results in modifying the visible light, which is really the chief object of concern to the ophthalmologist. There is a close kinship in the absorption of most of this recent crop of glasses. The prototype runs back nearly twenty years to the work of Fieuzal [119] who produced, specially with a view to protecting the eye against glare in the high mountains, a grayish green glass which cuts off the ultra violet very completely and shades down the blue so as considerably to shorten the spectral range of the rays transmitted. As one of us has already shown [18] the last line transmitted by this glass from the spectrum of the quartz arc is 404.6 $\mu\mu$ very faintly. Its absorption much resembles (loc. cit.) that of ordinary amber glass, except that the latter carries somewhat heavier absorption into the blue green accounting for its yellowish rather than greenish tinge. Either of these glasses is substantially as efficient as any of more recent origin in cutting out the ultra violet. In more recent times Hallauer [153] and Schanz and Stockhausen [309], have discussed at length glasses protective against the ultra violet and have brought out special protective glasses with this point in view, the former known by the name of the inventor, the latter under the trade designation "Euphos." At about the same period the firm of Rodenstock produced a glass of similar type under

the trade name of "Enixanthos" and various approximations to and imitations of these have from time to time appeared. A paper by Hallauer [155] gives the result of a rather thorough spectrographic study of all the protective glasses then in common use to which those interested in the subject may be referred. The composition of most of these glasses is held as an unnecessarily solemn secret, but it is generally understood that they are essentially iron-lead glasses. They run in color from a distinctly yellowish to a somewhat bluish green, varying in tone and transmissibility according to the composition of the various shades put on the market. An ordinary green glass bottle gives in the spectrograph about the same absorption as the medium densities of any of the glasses referred to. The deeper shades of any of them cut off the spectrum completely just about at the beginning of the ultra violet and weaken it well into the violet. The lighter shades transmit to wave lengths 360 $\mu\mu$ to 320 $\mu\mu$ with correspondingly less reduction in the general intensity. Any and all of them are completely effective against the abiotic radiations, even although the medium shades sometimes transmit very weakly in the region 3200 $\mu\mu$ to 3400 $\mu\mu$ as well as in the visible spectrum. To render the record of these recent protective glasses fairly complete there is shown in Plate 7 the iron arc spectrum and its transmission through the three ordinary grades of the Hygat glass of Rodenstock, an excellent type of this general group. Figure 6 is here the iron arc spectrum, 5, 4, and 3, respectively the light, medium and dark Hygat glasses, and Figure 1 a special protective glass of American manufacture designed specifically to reduce the red end of the spectrum beside cutting out the ultra violet and much of the violet and blue. In point of effectiveness in cutting out the ultra violet the differences between these various glasses of approximately the same shade are inconsequential and the choice between them lies mainly in the matter of taste as regards their particular color and absorption in the visible part of the spectrum. Of the recent glasses exploited in America the so called Noviol glass is remarkable for its extraordinarily sharp cut off of the spectrum in the blue.

Voege [388] in answering the question as to what spectral range of radiation gave the most satisfactory results held that the light from the clouds of a clear sky, being that light to which the eye through evolution had become adapted, was on the whole to be preferred. This would indicate the use of neutral glasses without selective absorption. Hertel and Henker [172] found that clouds and clear sky contain very little energy below 310 $\mu\mu$ and considered that the only

protection needed for artificial light sources was to reduce the light from them at the working distance to substantially the range of the sky spectrum. From these and other experiments they concluded that the best protective glass should preferably reduce the spectrum to approximately that of cloud or sky light. This again indicates the use of neutral non selective absorbing glasses. Against this view it may be properly objected that the eye in its evolution has rejected the whole infra red and ultra violet as ineffective and in fact derives very little useful illumination from the red at the one end and the violet and blue at the other. The luminosity values of these portions of the spectrum are very small and it may be added, fortunately very small, else the chromatic aberration of the eye would make distinct vision quite impossible. We are inclined, therefore, rather to the view that such radiation as produces the maximum required luminosity with the minimum energy access to the eye is best adapted to protect the eye from any and all injuries which may be due to excessive radiation. This indicates the use of glasses absorbing at both ends of the spectrum so as to bring the strongest light in the region of greatest luminosity, that is in the yellow green. As one of us has already shown [18] composite spectacles reducing the spectrum to a nearly monochromatic stripe in this region actually enable one to view the most powerful sources without discomfort while yet transmitting enough light to permit writing or reading one's notes. The glasses of Plate 7 in the deeper shades all show something of this characteristic absorbing both ends of the spectrum and in so far represent a slightly different type from the glasses which have preceded them. Crookes found that suitable absorption at both ends of the spectrum could not be obtained without encroaching somewhat on the visible portion but rendered this encroachment rather inconspicuous by using a heavy didymium glass which cuts out the yellow and leaves a pinkish tinge. This, however, is not an objection in cases requiring thorough protection unless the encroachment is so great as actually to be inconvenient in seeing. Where, therefore, practical protection against powerful sources of radiation is necessary, glasses meeting the requirement of maximum luminosity with minimum energy present material advantages. These advantages become practically inconsequential where the question is one of merely moderately reducing too bright general light, and the choice between such special protective media and ordinary neutral tint glass reverts again to a matter of taste.

ULTRAVIOLET LIGHT AS A GERMICIDAL AGENT.

Experimental Investigation of its Possible Therapeutic Value.*

F. H. VERHOEFF, A.M., M.D.

As is well known, light-waves of sufficiently short wave-lengths are highly germicidal to bacteria suspended in mediums which are transparent to these waves. The question has arisen, therefore, whether or not it may be possible to make use of ultraviolet light in the treatment of local infections.

Ultraviolet light has long been successfully used by Finsen in the treatment of certain skin diseases, notably lupus vulgaris, and recently has been employed by ophthalmologists in the treatment of vernal catarrh and trachoma, also, it is asserted, with successful results. Its beneficial effect in these conditions, however, obviously is not necessarily due to a direct germicidal action, but possibly only to an irritant action on the tissues.

Since the cornea compared to other tissues of the body is relatively transparent to ultraviolet light, it follows that if it should prove impossible by this means to destroy bacteria within corneal tissue without at the same time producing undue injury to the tissue itself, the same negative results would be obtained in the case of all other tissues. For this reason the present investigation was confined to experiments on the cornea. These experiments were made in connection with an investigation by Louis Bell and myself on the effect of ultraviolet light on the normal eye, advantage being taken of the powerful light sources and apparatus therein employed.

Hertel [1], in 1903, reported the successful use of ultraviolet light in the treatment of corneal ulcers, asserting that here it had a direct germicidal action on the infecting bacteria. He also made some interesting experimental observations in this connection, the most important one from a therapeutic point of view being that he was able to abolish the motility of cholera bacilli enclosed in a quartz cell and

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* Reprinted by permission from the Journal of the American Medical Association, March 7, 1914, Vol. LXII, pp. 762–764.

1 Hertel, E.: Experimentelles über ultraviolettes Licht, Ber. ü. d. 31 Vers. d. ophth. Ges., Heidelberg, 1903, p. 144.

placed within the anterior chamber of a rabbit's eye, by exposing them to the action of ultraviolet light passing through the cornea. The source of the light was a magnesium electrode giving off rays with wave-lengths from 0.28 to 0.309 microns, and the exposures were from twenty-five to thirty minutes. The current was from 3 to 4 amperes. Later he obtained the same result with his cadmium-zinc electrode. Hertel assumed that the bacteria were actually killed, but he did not state that this was demonstrated by means of cultures. He also did not exclude the possibility that the effect on the bacilli was due to heat.

Hertel, in addition, tested the therapeutic action of ultraviolet light on a series of rabbits' eyes in which he had produced staphylococcic corneal ulcers and obtained "pleasing results." The resulting scars were slight and no changes could be found in the depths of the eyes. These results, however, it seems to me, lose any possible significance when it is considered that staphylococcic corneal ulcers artificially produced in rabbits as a rule promptly heal without any treatment, as I have frequently observed.

Hertel maintains that light of short wave-lengths has a greater deleterious effect on bacteria than on tissue-cells. This may be true for very short waves, but it is certainly not true for waves which are able to pass through the cornea. Thus, I found that severe keratitis could be produced by exposing the cornea through a crown screen to a quartz mercury-vapor lamp at a distance of 20 cm. for one and one-half hours, whereas staphylococci suspended in distilled water and exposed under the same conditions were not killed in six hours[2]. This experiment also would seem almost alone sufficient to prove the impossibility of destroying bacteria within the clear cornea without producing too much injury to the corneal cells. This being the case, it is almost inconceivable that bacteria could be destroyed in a cornea infiltrated with pus-cells and so made practically impassable to germicidal waves.

Hertel also attached importance from a therapeutic point of view to the conjunctival hyperemia and cell irritation produced by ultraviolet light. The practical value of these factors is questionable, and the latter factor would seem more likely to do harm than good in the case of corneal ulcers in which the cells already have sufficient un-

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2 It is important to note that for these long exposures it is necessary to keep the bacterial container surrounded by cool water, as otherwise the bacteria may be killed in an hour or so by the accumulated heat. The lamp and screen used in this experiment are described later.

favorable influences to contend with. In any case they would not justify the use of ultraviolet light in treatment of such conditions in the absence of any germicidal effect of the light.

In a later communication, Hertel[3] reported in detail a series of clinical cases of corneal ulcer treated by means of ultraviolet light from a cadmium-zinc electrode. The latter he recommended as being equal in efficiency to the magnesium electrode and at the same time more practical to use. Twenty-six cases were treated with light therapy alone, 8 cases with light therapy followed by Saemisch section, and 13 cases with light therapy followed by cauterization or the latter and Saemisch section. Thus in 21 out of his 47 cases of corneal ulcer, the result of the light therapy was so unsuccessful that cauterization or Saemisch section had to be undertaken. These results do not seem impressive for an agent that is supposed to kill the bacteria within the ulcers. Hertel exposed his patients from three to five minutes two or three times daily. At the most this was equivalent to a daily total exposure of only fifteen minutes. Now he had found that it required from twenty-five to thirty minutes to kill (or inhibit)? bacteria exposed through a perfectly clear cornea. How then could it be expected that an exposure of fifteen minutes would suffice to kill them in a purulent infiltrate which acts as a far more effective barrier to ultraviolet light?

In a communication to appear later, Louis Bell and I show that interrupted exposures to ultraviolet light with intervals of less than twenty-four hours have practically the same effect on the cornea as a continuous exposure of the same total length. For this reason by frequently repeating his exposures, Hertel undoubtedly increased the injury to the corneal tissue without at the same time, in all probability, obtaining a corresponding increase in germicidal action.

In his experiments and in the treatment of his cases Hertel employed no screens. Thus the cornea had not only to contend with the rays that could penetrate it, but also with those stopped within the stroma and at the surface. As the rays stopped near the surface are evidently useless so far as killing bacteria within the stroma is concerned it occurred to me that by screening them out and so decreasing the damage to the cornea, longer exposures might safely be used, thereby increasing the possibility of a germicidal effect within the cornea. The screen selected for this purpose was a crown glass, which permitted

[3] Hertel, E.: Experimentelles und klinisches über die Anwendung lokaler Lichttherapie bei Erkrankungen des Bulbus, Arch. f. Ophth., 1907, lxvi, No. 2, p. 275.

only waves greater than 0.295 microns in length to pass[4]. As will be seen, however, this procedure was unsuccessful. No germicidal effect on bacteria within the cornea could be noted even when exposures through this screen were used which were sufficient to produce severe keratitis and even injure the epithelium of the lens capsule.

As light sources in the following experiments the magnetite arc and the quartz mercury-vapor lamp were chiefly used. To obviate the remote possibility that the cadmium-zinc arc employed by Hertel might possess some special advantage, this arc was also used. That greater intensity was obtained with our cadmium-zinc arc and quartz lens than was obtained by Hertel is proved by the fact that not only severe keratitis but also marked changes in the epithelium of the lens capsule were produced.

The mercury-vapor lamp used was the Cooper-Hewitt model without the globe (220 volts, 3.5 amperes).

The magnetite arc was of the ordinary self-regulating type as known to trade, without the globe. The voltage was about 80, the amperage from 9.8 to 10. The light was passed through a quartz water-cell 5 cm. in thickness, and concentrated on the cornea by means of a quartz lens 4 cm. in diameter and 9 cm. in focal length, placed 20 cm. from the light source. In Experiments 6 and 7 still greater intensity was obtained by means of a second quartz lens 23 mm. in diameter and 15 mm. in focal length.

In the case of the cadmium-zinc arc, the same apparatus was used except that the electrode consisted of an alloy of equal parts of cadmium and zinc in a thin-walled copper cylinder, and was water-cooled. The water-cell was omitted. The voltage was about 80, the amperage about 6.8.

A number of experiments were first made by injecting staphylococci or pneumococci into the corneas of rabbits and after twenty-four hours exposing the resulting abscesses to the ultraviolet light. Healing did not seem to be hastened, but since recovery ultimately occurred, as it did also in the control eyes, these experiments are not regarded as sufficiently conclusive and are not given in detail. Experiment 1, however, in which tubercle bacilli were injected into the cornea, was

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[4] Since this wave-length has been found to be the limit of transparency for the cornea, it would be expected that such a screen would protect the cornea from injury, the longer waves not being absorbed by the latter. As a matter of fact I have found that it does almost completely protect the corneal stroma, but permits severe injury or destruction of the epithelium, corneal corpuscles and endothelium.

perfectly conclusive since the resulting lesions continued to progress in both eyes alike. In the other experiments the exposures were made immediately after the injections, that is, with the corneas clear, so that the conditions were the most favorable possible for germicidal action of the light. The results in these experiments, moreover, are clear-cut, because if the light had killed the bacteria, abscesses would not have formed. This is proved by Experiment 2, in which in the control eye the bacteria were first killed by exposure to ultraviolet light before they were injected into the cornea.

Experiments.

Experiment 1. April 10, 1912, a suspension of virulent tubercle bacilli is injected into each cornea of a rabbit.

May 1, each cornea shows a small tubercle. The right eye is exposed to the quartz mercury-vapor lamp through crown screen $1\frac{1}{2}$ hours at 20 cm.

May 21, both tubercles have developed as usual. The animal is killed.

Experiment 2. June 22, 1912, a suspension of *Staphylococcus aureus* in distilled water is injected superficially into the left cornea of a rabbit. The remaining bacterial suspension is then exposed at 20 cm. for three minutes to the quartz mercury-vapor lamp. (Culture taken proves that all organisms have been killed.) This suspension of killed staphylococci is then injected into the right cornea of the same rabbit.

Each cornea is exposed to the quartz mercury-vapor lamp at a distance of 0.5 meter for fifteen minutes.

June 23, both eyes show marked photophthalmia. The left cornea shows well-marked abscess. The right cornea shows only a faint haze along the tract of the needle.

June 24, both eyes show increase in photophthalmia, with haze of corneal stroma and central loss of corneal epithelium. The abscess of the left cornea has increased in size and there is now hypopion. The right cornea shows no abscess. Enucleation is performed.

Experiment 3. Oct. 21, 1913, a suspension of *Staphylococcus aureus* in distilled water is injected into each cornea of a rabbit, the amount injected into the right cornea being three times that injected into the left. The left eye is then exposed for thirty minutes to the

cadmium-zinc arc through a quartz lens (no water-cell or screen of any kind being used).

October 22, the right eye shows intense inflammatory reaction, with a large abscess of the cornea and pus in the anterior chamber. The left eye shows equally intense inflammatory reaction and a corneal abscess about half the size of that in the right cornea. The abscess shows discrete points evidently corresponding to colonies of bacteria.

October 23, abscesses of the two corneas are now about equal in size, (3 mm. in diameter). The anterior chamber of each eye contains pus. Epithelium is entirely absent from the left cornea (this being confirmed by microscopic examination). The right cornea shows loss of epithelium only in the vicinity of the abscess. Enucleation is performed.

The lens capsule of the right eye after fixation in Zenker's fluid is examined in flat preparation and shows slight changes in the nuclei of the epithelium evidently due to the action of staphylococcus toxins, but no changes similar to those seen after exposure to ultraviolet light. The lens capsule of the left eye shows, in addition to these nuclear changes, well-marked changes characteristic of exposure to ultraviolet light — swelling and granular degeneration of the cytoplasm of the epithelial cells. Histologically both corneas present the same picture, and each contains numerous large masses of staphylococci.

Experiment 4. Feb. 8, 1913, a suspension of *Staphylococcus aureus* in distilled water is injected into each cornea of a rabbit. The left cornea is then exposed for thirty minutes to the cadmium-zinc arc *through a crown screen* and quartz lens, the image being kept on the injected area.

February 10, there are abscesses of equal size in the two corneas. The left eye in addition shows severe photophthalmia with marked general haze of cornea and large central loss of epithelium which includes area of abscess. This eye also shows exudate adherent to the posterior surface of the cornea behind the site of the abscess, due evidently to injury of the endothelium by the ultraviolet light. Enucleation is performed.

The lens capsule of the left eye, after fixation in Zenker's fluid, is examined microscopically in flat preparation, and shows marked changes characteristic of exposure to ultraviolet light.

Experiment 5. March 19, 1913, a suspension of *Staphylococcus aureus* in distilled water is injected into the cornea of a rabbit. The cornea is then exposed to the magnetite arc for forty-five minutes through the quartz lens, quartz water-cell, and crown screen. This exposure in the case of normal eyes had been found sufficient to cause

necrosis of the stroma cells and endothelium of the cornea, to cause hemorrhages in the iris, and to produce marked changes in the lens capsular epithelium.

March 20, there is abscess of the cornea. Marked photophthalmia is noted with loss of epithelium.

March 24, the abscess is larger. Enucleation is performed.

Lens capsular epithelium (flat preparation) on microscopic examination shows marked changes, and the iris shows numerous hemorrhages characteristic of exposure to ultraviolet light.

Experiment 6. Dec. 16, 1913, a suspension of *Staphylococcus aureus* in distilled water is injected into the cornea of a rabbit. The injected area is then exposed twenty minutes to the magnetite arc through a water-cell and a system of two quartz lenses. No screen is used.

As previously determined, with this arrangement an exposure of thirty seconds is sufficient to cause marked keratitis and destruction of the epithelium, while an exposure of twenty minutes causes complete destruction of the corneal corpuscles and softening and swelling of the stroma down to Descemet's membrane, and ultimately leads to vascularization and cicatrization of the cornea.

December 18, there is no abscess. Marked photophthalmia is noted. The cornea is hazy and epithelium is absent from three quarters of the surface of the cornea.

December 26, there is no abscess. The inflammatory reaction is almost gone. The cornea is softened and swollen.

December 29, the inflammatory reaction is increasing again (reaction of repair); the corneal tissue is very soft. Vascularization is well advanced.

Jan. 5, 1914, vascularization of the cornea is complete. The inflammatory reaction is subsiding.

January 9, the vessels are beginning to disappear. The cornea is leukomatous.

Experiment 7. Dec. 19, 1913, the suspension is injected and the exposure made as in Experiment 6, except that the time of exposure is six minutes. This exposure is sufficient to cause softening of the corneal stroma.

December 21, marked photophthalmia is noted. There is an abscess at the site of the injection.

December 23, the abscess is smaller. A culture is taken. Enucleation is performed.

Culture shows abundant growth of staphylococci. Lens capsular epithelium shows marked changes.

CONCLUSIONS.

The results of these experiments prove conclusively that ultraviolet light cannot under any conditions destroy bacteria within the cornea, even when the latter is perfectly transparent, without at the same time severely injuring the corneal tissue. Destruction of bacteria within the transparent cornea was obtained only when a light intensity and exposure were employed sufficient to cause complete destruction of the corneal corpuscles and intense injury to the corneal lamellae (Experiment 6).

Moreover, it does not seem possible that ultraviolet light could in practice be successfully used to destroy bacteria within a corneal abscess or ulcer, that is, when the cornea was no longer clear, even with the sacrifice of corneal tissue, as in the case of the actual cautery. For either the exposures would have to be impracticably prolonged, or such extreme intensity of light would be required that the heating effect would exceed that of the abiotic action. It is doubtful also if ultraviolet light of such intensity could be made available for therapeutic purposes.

It must be concluded, therefore, that so far as direct destruction of bacteria within any of the tissues of the body is concerned, ultraviolet light possesses no therapeutic value.

GENERAL CONCLUSIONS.

1. The liminal exposure capable of producing photophthalmia to the extent of conjunctivitis accompanied by stippling of the cornea, is in terms of energy about 2×10^6 erg seconds per square cm. of abiotic radiation of the character derived, for example, from the quartz lamp or the magnetite arc. About two and a half times this exposure, *i. e.*, 5×10^6 erg seconds per square cm. is required to produce loss of corneal epithelium.

3. The abiotic action of the cornea and conjunctiva produced by any radiating sources follows the law of inverse squares and is directly proportional to the total abiotic energy received. It can therefore be definitely predicted from the physical properties of the source.

3. After exposure of the eye to abiotic radiations there is a latent

period before any effects clinical or histological become perceptible. This period of latency in a general way varies inversely with the severity of the exposure, but a theoretical latency of 24 hours or more corresponds to an exposure entirely subliminal.

4. The combined effect of repeated exposures to abiotic radiations is equivalent to that of a continuous exposure of the same total length, provided the intermissions are not long enough to establish reparative effects. Approximately the exposures are additive for intermissions of somewhat less than 24 hours. Exposures of $\frac{1}{3}$ the liminal given daily begin to show perceptible effect after about 6 exposures. Daily exposures of $\frac{1}{6}$ the liminal repeated over long periods produce no effect whatever, except to give the external eye a degree of immunity against severer exposures. Actual abiotic damage to the external eye renders it temporarily more sensitive to abiotic action.

5. Abiotic action for living tissues is confined to wave lengths shorter than 305 $\mu\mu$, at which length abiotic effects are evanescent, while for shorter wave lengths they increase with considerable rapidity.

6. For the quartz arc and the magnetite arc the abiotic activity of the rays absorbed by the cornea is eighteen times greater than those which are transmitted by it. To affect any media back of the cornea requires therefore at least eighteen times the liminal exposure heretofore mentioned.

7. Even with exposures as great as one hundred and fifty times the liminal for photophthalmia the lens substance is affected to a depth of less than 20 μ, and this superficial effect undergoes in the rabbit complete repair. Such enormously intensive exposures, which we obtain with the magnetite arc and double quartz lens system may completely destroy the corneal epithelium, corpuscles, and endothelium. The corneal stroma may be strongly affected by waves shorter than 295 $\mu\mu$, which it completely absorbs, but is very slightly affected by the remaining abiotic radiation.

8. The histological changes produced by abiotic radiation are radically different from those produced by heat, and the cell changes are best seen in flat preparations of the lens capsule. The most characteristic change is the breaking up of the cytoplasm into eosinophilic and basophilic granules.

9. Changes in the lens epithelium like those following abiotic action, including the formation of a "wall" beneath the pupillary margin, are not exclusively characteristic of abiotic action, but may be produced by ordinary chemical reagents. They are, therefore, characteristic not of abiotic action alone, but of chemical action in general.

10. Abiotic radiations certainly do not directly stimulate, but on the contrary apparently depress mitosis. Their action in this respect also is materially different from that of heat.

11. The lens protects completely the retina of the normal eye even from the small proportion of feebly abiotic rays which can penetrate the cornea and vitreous humor.

12. Experiments on rabbits, monkeys and the human subject prove that the retina may be flooded for an hour or more with light of extreme intensity (not less than 50,000 lux), without any sign of permanent injury. The resulting scotoma disappears within a few hours. Only when the concentration of light involves enough heat energy to produce definite thermic lesions is the retina likely to be injured.

13. The retina of the aphakic eye, owing to the specific and general absorption of abiotic radiations by the cornea and the vitreous body, is adequately protected from injury from any exposures possible under the ordinary conditions of life, even without the added protection of the glasses necessary for aphakic patients.

14. To injure the cornea, iris, or lens, by the thermic effects of radiation, requires a concentration of energy obtainable only under extreme experimental conditions.

15. Infra-red rays have no specific action on the tissues analogous to that of abiotic rays. Any effect due to them is simply a matter of thermic action, and such rays are in the main absorbed by the media of the eye before reaching the retina.

16. Actual experiments made on the human eye show conclusively that no concentration of radiation on the retina from any artificial illuminant is sufficient to produce injury thereto under any practical conditions.

17. Eclipse blindness, the only thermic effect on the retina of common occurrence clinically, is due to the action of the concentrated heat on the pigment epithelium and choroid, this heat being almost wholly due to radiations of the visible spectrum within which the maximum solar energy lies.

18. The abiotic energy in the solar spectrum is a meagre remnant between wave lengths 295 $\mu\mu$ and 305 $\mu\mu$, aggregating hardly a quarter of 1% of the total. At high altitudes and in clear air it is sufficient to produce slight abiotic effects such as are noted in snow blindness and solar erythema, the former only occurring with long exposures under very favorable circumstances and the latter being in ordinary cases complicated by an erythema due to heat alone. The amount

of abiotic energy required to produce a specific effect in solar erythema is substantially the same as that required for mild photophthalmia.

19. Erythropsia is not in any way connected with the exposure of the eye to ultra violet radiations, but is merely a special case of color fatigue, temporary and without pathological significance.

20. Vernal catarrh and senile cataract we can find no evidence for considering as due to radiations of any kind.

21. Glass blower's cataract often charged to specific radiation, ultra violet or other, we regard as certainly not due to ultra violet light but probably due to the overheating of the eye as a whole with consequent disturbed nutrition of the lens.

22. Commerical illuminants we find to be entirely free of danger under the ordinary conditions of their use. The abiotic radiations, furnished by even the most powerful of them, are too small in amount to produce danger of photophthalmia under ordinary working conditions even when accidentally used without their globes. The glass enclosing globes used with all practical commercial illuminants are amply sufficient to reduce any abiotic radiations very far below the danger point.

23. Under ordinary conditions no glasses of any kind are required as protection against abiotic radiations. The chief usefulness of protective glasses lies not so much in their absorption of any specific radiations, as in their reducing the total amount of light to a point where it ceases to be psychologically disagreeable or to be inconveniently dazzling. Glasses which cut off both ends of the spectrum and transmit chiefly only rays of relatively high luminosity, give the maximum visibility with the minimum reception of energy. For protection against abiotic action in experimentation, or in the snow fields, ordinary colored glasses are quite sufficient.

24. So far as direct destruction of bacteria within the cornea or any other tissues of the body is concerned, abiotic radiations possess no therapeutic value. This is due to the fact that abiotic radiations that are able to penetrate the tissues are more destructive to the latter than to bacteria.

SYSTEMATIC REVIEW OF THE LITERATURE RELATING TO THE EFFECTS OF RADIANT ENERGY UPON THE EYE.

By C. B. Walker, A.M., M.D.

Chronological Account.

Historically we find the first study of the properties of the ultra violet light in connection with the eye was commenced long before high power lamps were invented or the rôle of ultra violet rays in producing eye-injuries was established. The first work, was stimulated by purely scientific interest with no prophylaxis or therapy in view; in fact these latter factors did not enter for several decades.

As early as 1845, Brucke [58] laid the foundations for subsequent work, in his investigation of the reason for the invisibility of the ultra violet rays. In order to determine whether these rays failed to traverse the eye media or failed to stimulate the retina, he first studied the absorptive power of the eye media. He found that Gum Guaiacum had a characteristic bluish appearance in ultra violet light. By means of this substance he was able to say that the lens absorbed ultra violet rays strongly and the cornea and the vitreous humor to a less extent. Later with the assistance of Karstein he found with sensitive paper that a combination of lens, vitreous humor and cornea, diminished somewhat the intensity of the violet, began to absorb more just outside the visible spectrum, was especially strong on the "M" (372 $\mu\mu$) line of the Draper spectrum, and practically total beyond, that is for rays less than 370 $\mu\mu$.

In 1852 Stokes [357] discovered fluorescence and thus afforded another means of studying the absorption of the eye media. Donders [97] with Rees in 1853 threw a solar spectrum upon a screen covered with quinine sulphate which by its fluorescence rendered the ultra violet rays visible. Various eye media, unfortunately enclosed in glass containers, were then interposed in the path of the ultra violet rays. The absorption power of glass itself rendered these results of little value.

In 1855 Helmholtz [161] studied the lower limit of the visible spectrum using a quartz prism, but his high-grade myopia interfered. He also used the fluorescing screen of quinine sulphate and studied the fluo-

rescence of the crystalline lens, of various solutions and of the retina. The fluorescence of the latter he discovered in the morbid state with Setschenow. A number of observers, Eisenlohr [106] in 1856, Janssen [198] in 1860, Franz [121] in 1862, Listing [228] in 1865, Mascart [240] in 1869, Sekulic [334] in 1872 and Sauer [305] in 1875 continued the study of the same question of the length of the visible spectrum in much the same manner. Their results, with the exception of those of Eisenlohr and Sauer, added little however, since, as the latter pointed out they were not free from certain objections which will be taken up more in detail under the discussion of the properties of the lens.

As early as 1858, Charcot [65] gave the first description of photophthalmia and erythema produced by a small electric laboratory furnace (cf. page 635).

In 1867 Czerny [85] made the first experimental observations on the effect of direct sunlight on the retina. Even through heat filters he found he could produce in the rabbit's eye marked destruction of retinal elements in 10 to 15 seconds exposure with concentrated sun rays. Deutschman [89] later (1882) showed that these changes could be noted in 1 second in the same manner. Herzog [176] confirmed these results in 1898 and reduced the exposure time to $\frac{2}{5}$ secs. In some of these cases there were cataractous lens changes but no outer eye disturbance.

Tyndall [370] in 1876, made an important contribution to the subject in establishing the fact that ultraviolet rays are absorbed by the atmosphere, since the ultra violet content of the solar spectrum was found to be much greater on high mountains than on low plains.

In 1806 [418] Wenzel and later von Beer in 1817 [184] pointed out the disposition to cataract among glass blowers.

The introduction of the arc light in 1879 and 1880 mark an epoch in the history of this subject, for at least two reasons; first, because a means was afforded for far more accurate and extensive study of ultra violet light which the electric arc so abundantly supplies; secondly because immediately after the use of the electric arc for lighting and high temperature furnaces became general, cases of what was later designated as ophthalmia electrica, began to make their appearance. Martin [237] and Nodier [258] in 1881 reported on these cases, and although they correctly described the symptom complex, their explanation of the phenomenon as a sympathetic reflex from the injured retina was soon proved to be incorrect.

In 1882 Leber [221] after studying the cases of cataract formation after exposure to lightning came to the conclusion that such cataracts were produced electro-chemically.

In 1883 de Chardonnet [67] used the ultra violet rays from the arc light to study the absorption power of various parts of the eye, and emphasized for the first time the very important rôle of the lens as a determinant for the limit of visibility of the short waves of the spectrum, by virtue of having the highest absorption power of all the eye parts. He argued therefore that aphakic eyes should have a greater range of spectral vision than normal eyes. Accordingly he examined two patients who had clear eye media after the extraction of cataractous lenses. He asked these patients to observe an arc light through a quartz glass plate, thinly silvered so as to cut out all rays except those between 343 $\mu\mu$ and 301 $\mu\mu$ lines. Normal eyes could not make out on looking through this glass whether the arc light was burning or not, but the aphakic patients could even detect motion of the light.

In 1886 Meyhöfer [245] found 11.6% of glass blowers under 40 years of age had cataracts, and these were mostly left sided where heat exposure was greatest.

In 1888 Hess [177] allowed an electric spark to impinge on the supra-orbital region of a rabbit and produced equatorial cataracts. There was more or less central distruction of lens capsule and vacuolization of anterior lens fibres with peripheral increase of mitotic figures in the capsule. This result was later confirmed by Chiribuchi, but was shown to be an electrochemical rather than abiotic effect.

It remained for Widmark [415] in 1889 to experiment with the effect of ultra violet light on the eyes of the laboratory animals. He reproduced the stages of electric ophthalmia in the rabbits' eyes and considered the reaction to be of the nature of an inflammatory erythema. He first demonstrated the protective power of the lens by interposing a fresh rabbit's lens in the path of the ultra violet rays to which the rabbit's eye was exposed. The rabbit's eye in this case failed to give the characteristic reaction.

Hirschberg [172] in 1898 first suggested the possible influence of intense sunlight in producing early senile cataracts in India and in the country, though Schulek [329] had in 1895 from the statistics of Grosz, already noted that the senile cataract was more common in people working on the hot plains than in city dwellers. Schwitzer [332] was the first to incriminate the ultra violet portion of the sunlight as an etiological factor in these cases. Hirschberg [184] in 1901 first noted that the senile cataract almost always began in the lower quadrant of the lens.

Perhaps stimulated by the possibilities of protection to the eyes suggested by Widmark's experiments, Schuleks [345] in 1900, examined a

great number of substances for protective properties. Unfortunately the substances he found to have the necessary transparency and absorptive power were certain liquid solutions. His results for the absorptive power of the lens and the other parts of the eye were the same as those obtained by de Chardonnet. The study of protective glasses was later taken up by Staerkle[348], and Vogt[396], and quite recently with more success by Hallauer[154a155], Schanz and Stockhausen[312], and Birch-Hirschfeld[31].

Widmark[318] in 1901 and 1902 continued to develop the experimental method of studying the problem on the eyes of laboratory animals. He introduced some very ingenious experimental arrangements and was the first to show with the aid of the microscope that ultra violet rays can produce definite pathological lesions of the corneal and lens epithelium as well as of the conjunctiva and the skin of the lids and face. Further he believed he had ascertained that the injuries to the lens can be readily aggravated until cataract formation is the result. He found that heavy glass (18 mm. thick) when interposed prevented these changes. Solutions of quinine sulphate were equally protective. He was the first to note the similarity of ophthalmia electrica and the outer eye trouble in snow-blinding.

It was not till 1907 that these results received some confirmation by Hess[179]. A number of observers had looked for lens changes both before and afterwards without success, or with variable results. Thus Ogneff[259] in 1896 using an arc light of 5000 to 8000 c. p. noticed no lens trouble but much outer eye trouble as Widmark[415] had shown in 1889. Herzog[176] in 1898 repeated this work with a heat filter and a common glass optical system on young rabbits and considered that any small effect such as he found was due to heat transformation. Birch-Hirschfeld[30] in 1904–5 found no lens changes with 4–10 min. exposure to a 4 amp. Finsen light. Hertel[350] in 1903 using the magnesium spark likewise noted no lens change; nor did Strebel[175] using 5 min. exposures to a 6 amp. iron arc light.

Hertel's[175] work in 1903 was based on his idea that the pathogenic range of ultraviolet rays should be determined on the living cell rather than on the photographic plate since there was a difference in the action of these rays on chemical and living substance. He therefore enclosed certain bacilli, in tiny quartz glass boxes which could be inserted into the aqueous or vitreous chambers of the eye. Exposing the eye, then, to the ultra violet rays from a magnesium electrode spark, he found that waves of 280 $\mu\mu$ would not pass through the lens and kill the organisms (B. Coli) behind it even after 60 min.

exposure, while bacteria in the anterior chamber were killed in 25 to 30 min. but not in the control with common glass interposed (cf. page 749). In none of the eyes, 26 in all, was lens trouble noted.

In 1904 definite pathological changes believed to be due to ultra violet light were noted by Birch-Hirschfeld [38] in the finer structures of the retina.

In 1906 and 1907 Vogt [395] and Hallauer [153] began the careful study of transparent and colorless protection-glasses, and the latter produced by a secret process the so called "Hallauer glass."

In 1907 Schanz and Stockhausen [309] also invented and patented a new glass, which they called "Euphos glas."

In 1908 Birch-Hirschfeld [35] studied in five cases visual field changes produced by uviol lamps, showing sector and ring formed scotomata for red and green to be the predominant varieties, but later (1912) he objected [41] to the ringscotoma found by Jess [200] in the same year.

Voege [388] in 1908 asserted that daylight might be taken as the ideal light, especially "cloud light." He compared the spectra of various high power lights protected with milk and opal glass coverings, with the spectra of cloud light and found them to compare favorably, and therefore concluded that these lights so protected are not to be considered dangerous when properly used.

In 1909 Schanz and Stockhausen [316] vigorously opposed this attitude and their view was supported in the same year, by the appearance of the statistical study of Handmann [157] showing that the senile cataract begins in the region of the lens most exposed to the short wave length light of the sky, that is in the lower half.

In this year Birch-Hirschfeld [38], Schanz and Stockhausen [316], and Hallauer [152] (on human lens only), by spectrophotographic method, measured with the greatest care, the absorptive power of various kinds of glass, the cornea, vitreous humor and lens of various animals and of the human eye. They also made careful measurements of the spectral range of a great variety of light sources with and without covering of common glass, milk glass and opal glass.

In 1910 Schanz and Stockhausen [318] made a very careful study of the fluorescence of the human lens by a hitherto unused method, and also examined more carefully than before the spectrum of the glass blowers' furnace and the conditions under which the glass blowers were forced to work. They contended that the glass makers' cataract is due the longer of the ultra violet rays with perhaps the assistance of the shortest visible rays.

Also in 1910 Hertel and Henker [172] accepting Voege's idea that the

ideal light is cloud light or skylight, used the most accurate instruments available in the laboratory of C. Zeiss, in Jena, to measure the percentage absorptive power at different points in the spectrum, of various glasses. These glasses were all found to be inferior to opal and milk glass for the purpose of enclosing strong arc lights to produce a spectrum most nearly approaching in quality and quantity, the spectrum obtained from sky light.

Recently (1912) Martin [238] has verified some of the results of Widmark, Hess, and Romer, while Carl Behr [17] has reported some very interesting functional disturbances of light adaption power of the eyes in patients working by artificial light. These results will later be taken up more in detail.

Having thus rapidly traced the important steps in the progressive development of the knowledge of ultra violet light in relation to the eye, the mass of findings may doubtless be rendered much more available by considering them separately and in more detail with reference to the various parts of the eye.

The Outer Eye.— Photophthalmia, Vernal Catarrh.

Probably ophthalmologists have experienced less difficulty in reaching definite conclusions, concerning the condition called ophthalmia electrica or photophthalmia (Parsons [265]), than with any of the other effects of ultra violet light. As to the symptom complex little has been added since the first report in 1858 (cf. page 635) of Charcot [65] and the later observations of Martin [238] and Nodier [258] in 1881, shortly after the general introduction of the electric arc for lighting and furnaces, (in 1879 and 1880). The workmen most exposed to these arcs, particularly the furnace arc, began to complain of symptoms that we now know to be due to photophthalmia (see page 634). In a week the eyes were practically normal. The affection of the surrounding skin known as dermatitis electrica was not unlike that of sunburning of severe grade except in its origin. As in sunburn, a tanning was notable after the inflammation had subsided, for several weeks. Although retinal changes were seldom noted with the ophthalmoscope, functional disturbances were observed such as temporary blindness or scotomata, floating spots of red, yellow or blue or occasionally erythropsia, or red vision. Therefore the very early writers were inclined to believe the outer eye trouble followed sympathetically from the retinal injury. These retinal disturbances also led Terrier [363]

in 1888 to divide the large number of reported cases into two classes; a mild group without retinal disturbances and of good prognosis, and a severe group with retinal disturbances and of bad prognosis. However, after the classical experiments of Widmark these theories and classifications were no longer found to be useful. Widmark [415] in 1889 exposed the rabbit's eye to various parts of the arc light spectrum. He found that when a 1200 c. p. arc light was used for 10 min. on the rabbit's eye without screening out any ultra violet rays all the typical symptoms of electric ophthalmia appeared after a latent period of 6 hours. By varying the time of exposure any degree of injury could be produced from a mild erythema to ulceration of the conjunctiva and cornea. But if a common glass plate 0.5 to 1.0 cm. thick was interposed the rabbit was entirely protected. Thus he established for the first time that rays below 300 $\mu\mu$ in length were chiefly responsible for the outer eye trouble. This particular point was confirmed by Ogneff [259], Hess [177], Kiribuchi [203] and subsequently by practically all observers. Further Widmark concluded that ultra red and the visible rays are entirely without effect, outside of common heating effects.

Widmark made another important contribution to the knowledge of this subject when he drew attention to the striking resemblance of ophthalmia electrica and the disturbance found on the outer eye in cases of snow-blinding. He showed that they both had the same latent period and were ushered in with the same syndrome of symptoms. Further that erythropsia, temporary blindness, or blind spots occurred in both. The fact that ultra violet light is stronger on high mountains and snow covered surfaces as had been shown by Tyndall and subsequently verified by Helmholtz, Langley, Cornu and Mascart, was a further argument emphasized by Widmark in support of his contention that ultra violet rays are responsible for both ophthalmia electrica and snow blinding.

Birch-Hirschfeld, Hertel, Best and others had up to 1907 found a thick plate of common glass to be sufficient protection from electric ophthalmia as Widmark had pointed out. But in 1907 Stockhausen after one half hour working with arc lights received a severe ophthalmia electrica through glass protection. Schanz & Stockhausen [310] therefore repeated Widmark's experiment and found common glass to be inefficient protection for long intense exposure. They were able to produce the characteristic symptom in a rabbit's eye through 18 mm. of common glass after 4 hours' exposure to a 15 amp. arc light. Thus stimulated they studied the manufacture of glass carefully,

and finally produced the yellowish colored "Euphos glas" which they recommend as very satisfactory, not only for protection glasses, but also for use in making arc light coverings or mantles.

However Birch-Hirschfeld [34] in 1907 considered the above exposure so intense as to afford no criterion for cases as they usually occur. From his results he asserted that one need not be afraid to use the ordinary smoked, uviol, flint, or even common glass, in the great majority of cases. He exposed a rabbit's eye for 1 hour as close as 10 cm. to a uviol lamp (mercury vapor tube) protected only by a 2 mm. thickness of common glass. After the 6 hour latent period no symptoms whatever developed although the control rabbit's eyes were badly damaged. Further he exposed his own eyes to a 3000 c. p. quartz mercury arc lamp at 1 meter distance using smoked glass goggles as a protection. Although the surrounding skin of his face was burned, no symptoms of ophthalmia electrica developed. Birch-Hirschfeld [34] also proved that by daily exposure of the rabbit's eye to ultra violet rays a chronic inflammation of the outer eye could be produced which was very similar in appearance, both grossly and histologically, with vernal catarrh, originally considered by Schiele [233] in 1899 to be a result of exposure to the light rays of the sun. In this investigation Birch-Hirschfeld exposed the rabbit's eyes for 10 min. every day for 180 days at a distance of 10 cm. from the "Uviol lampe" of Schott. The eye lids of the rabbits were everted during the exposure. After passing through the usual acute ophthalmia electrica a chronic inflammation was established similar to vernal catarrh, but no trouble in the lens or retina was noted.

Birch-Hirschfeld considered rays shorter than 330 $\mu\mu$ to be primarily responsible for the outer eye disturbance in this case, still rays from 330 $\mu\mu$ to 400 $\mu\mu$ or more could not be excluded as etiological factors. However he agreed with Axenfeld and Ruprecht [9], 1907, that these factors could not entirely explain vernal catarrh. Vogt [401] in 1912 thought that exacerbations at least in the disease depended on thermic influences.

The Cornea: — Absorption, Injuries.

That the cornea might suffer severe injury in bad cases of ophthalmia electrica was early noted by Terrier [363] in his report of 1888. In these cases a dull haziness of the cornea with perhaps a phylectenular condition or bleb formation was first noted. This condition could either go on to ulcer formation by infection or to panuus formation

by vascularization. The ulcer formation as is usually the case, often lead to, or was accompanied by, iritis. Corneal disturbance in snow blinding has been occasionally reported. Hildige [191] in 1861 and Reich [184] in 1880 saw small ulcers.

Widmark [415] in 1889 studied the progress of the earliest changes on the cornea due to ultra violet rays. With the aid of the microscope he found first in the corneal epithelium a swelling and necrosis of the nuclei leading to necrosis of epithelial cells, and small areas of desquamation followed sometimes by ulcerative conditions and usually by opacities. These findings were at once verified by Ogneff [259] and Bresse [59] and later by many others. Hertel [167] in 1903, repeating this experiment and with rays of 309 $\mu\mu$ to 280 $\mu\mu$ from the magnesium spark, was able to produce the same corneal injuries, as well as to kill, or at least demoralize bacilli enclosed in quartz containers and placed in the anterior chamber. This could not be done when common glass was interposed in the control experiment. That this fact may be taken as evidence that rays of 280 $\mu\mu$ were able to penetrate the cornea does not follow, was pointed out two or three years later by Birch-Hirschfeld, Schanz and Stockhausen, who considered that rays of greater length than 280 $\mu\mu$ in sufficient amount to kill organisms could not be excluded (cf. page).

Widmark made no attempt to determine the absorptive power of the cornea by spectrophotographic methods. Schanz and Stockhausen [312] were among the first to attempt accurate measurements in this way on the human as well as on the animal cornea. They found that all rays below 300 $\mu\mu$ are absorbed by the cornea. Hess, Birch-Hirschfeld and Herzog verified this measurement and again later Birch-Hirschfeld [32] attempting still greater accuracy, with the same method, placed the absorptive limit at 306 $\mu\mu$. Parsons [266] in England in the same way found rays above 295 $\mu\mu$ able to penetrate the cornea.

Still later, in 1909 Schanz and Stockhausen reconsidered the limit of 300 $\mu\mu$ for the absorptive power of the cornea placing it at 320 $\mu\mu$ for all practical purposes, since the spectrum was so weakened between 320 $\mu\mu$ and 300 $\mu\mu$ as to be without action on the lens. 300 $\mu\mu$ was however still considered the point of complete absorption.

Martin [238] in 1912 agreed with Parsons that the cornea offered no resistance to waves above 295 $\mu\mu$ length but all beyond this limit were completely cut off.

The Aqueous and Vitreous Humors.

The humors of the eye seem to be the most silent regions as far as response to insult from ultra violet light sources is concerned. Since they have an absorptive power, never greater, and often less than that of the cornea, the latter apparently protects them from the action of the injurious rays.

Donders [97] who made the first attempt to measure the absorptive power of vitreous humor alone was not aware of the fact that the containing vessel must be made of thin quartz glass, so that his results were of no value. After him Soret [345] in 1879 reported the first reliable results. He found the vitreous humor able to absorb rays of lengths less than 294.8 $\mu\mu$ in thicknesses of 1 cm. and still smaller values for thinner layers. The values of de Chardonet [67] in 1883 were still lower. He found the absorptive value to lie between the 310 $\mu\mu$ and 304 $\mu\mu$ lines.

Birch-Hirschfeld [34] in 1909 found that the vitreous humor in 1 cm. layers has an absorptive power, practically constant for all animals, of rays less than 300 $\mu\mu$ thus being the same as common glass. Schanz and Stockhausen [309], Vogt [396], Hess [178], Ogneff [259], Birch-Hirschfeld [31] and all recent observers have also confirmed this value for 1 cm. layers of vitreous humor. Parsons [266] for thinner layers, —$\frac{3}{16}$ of an inch,— found absorption to begin at 280 $\mu\mu$ and become complete at 270 $\mu\mu$. Martin [238] in 1912 confirmed the later results and found no change in the absorptive power of eye media as long as 8 hours after death.

The Iris.

The iris and uveal tract have long been noted to suffer in severe exposures to short wave lengths. Martin [238] and Nodier [258] in 1881 noted inflammation of the iris in severe cases, confirmed by Terrier [363] in 1888. The very short exposure with light rays by Czerny [85] in 1867, Deutschman [89] in 1898 and Herzog [176] in 1898 gave no notable iris changes beyond slight hyperaemia. Hess [177] in 1888 by use of the electric spark impinging in the supraorbital region, and Kiribuchi [203] in 1900 with the Leyden jar spark were both able to produce marked uveitis. Gardner [184] in 1871, Berlin [22] 1888, and Ewald [181] 1891, have reported hyperemic and swollen iris in snow blinding.

Widmark [415] in 1889 noted microscopically in cases of 2–4 hours

exposure a marked swelling and hyperemia of the ciliary body, and in later experiments in the same way noted small hemorrhages in the iris. Gross examination showed myosis and discoloration of the iris. Ogneff [259], Terrier [363] and Weiss [31] confirmed these findings.

Birch-Hirschfeld [37] in 1904 with the Finsen 3.5 to 4.5 amp. arc light for 5–10 minutes noted iritis and cyclitis in 6–12 hours with fibrinous exudate into the anterior and posterior chambers. Further experiments in 1908 with the Schott lamp 10 min. exposures at 10 cm. daily for 180 days showed practically no effect on the iris though a chronic conjunctival inflammation was produced.

Martin [238] in 1912 noted, in rabbits exposed 1½ to 2 hours at a distance of 1 in. from a Kromayer mercury vapor lamp hyperemia and myosis of the iris but no exudates. Further by the hemolytic method of Romer he found that the iris showed evidence of damage with intensities above 1 hour exposure at 4 in. distance. Whether the injury to the iris produced when the light is of sufficient strength is a direct result of light rays or a secondary effect of the corneal and outer eye injury was not made clear.

The Lens: — Absorption; Fluorescence, as a Determinant of Visibility of ultra violet Rays; Injuries; Cataracts.

The reports of different observers upon the absorption power of the crystalline lens have varied considerably. Birch-Hirschfeld [37] in 1909 accounted for the long list of previous variations in the following manner. Aside from the personal equation or individual variation in observation, there is a considerable variation in the absorptive power of lenses of different animals of the same species as well as of different species. His results may be tabulated thus:

Animal	Average absorption	Range of Variation in Different Animals
Swine	330 $\mu\mu$	15 $\mu\mu$
Calf	328 $\mu\mu$	12 $\mu\mu$
Ox	385 $\mu\mu$	30 $\mu\mu$ increasing with age.

To a less extent thickness plays a part, though not so very great, since 5 mm. of rabbit lens has about the same absorptive power as 10 mm. of ox lens, for waves less than 390 $\mu\mu$. The formula for the

effect of thickness or intensity before and after transit shows a variation possibility of small degree thus,—

$$J^1 = J_0 \cdot e^{kd} \quad \text{where } J_1 = \text{intensity after transit}, \; J_0 = \text{intensity before transit}$$

d = thickness and k = coefficient constant.

The human lens he found to vary considerably, as will be shown later, with such factors as age, consistency and color.

As has been stated the absorptive power of the lens and other eye media for ultra violet rays, and the limit of visibility of the spectrum in the ultra violet region are two problems whose investigation has been carried forward in the same stages since it was obvious from the start that the determination of one would throw much light on the other.

Brucke [58] really opened the subject in 1845 when he speculated as to the range of the visible spectrum and the reason for the invisibility of the ultra violet rays. In the manner described he found the ox lens to absorb rays below 370 $\mu\mu$. Donders [97], using the method of fluorescing screens of quinine sulphate, discovered by Stokes, attempted to measure the absorptive power of the lens but the glass containers vitiated his results.

Stokes [357] by direct observation of the solar spectrum through a quartz prism, thought he could see as low as the 372 $\mu\mu$, 358 $\mu\mu$, and 335 $\mu\mu$ lines and perhaps further.

In the same way Helmholtz [161] with a quartz optical system could see a few lines in the 372 $\mu\mu$ and 318 $\mu\mu$ region, although his eyes were very myopic. He also observed the ultra violet rays directly through holes in the fluorescing region of a screen of quinine sulphate upon which the spectrum was thrown.

By similar methods Listing [228] placed the limit of visibility at the 372 $\mu\mu$ line and Sekulic [334] at the 358 $\mu\mu$ line. Mascart [240] however, using high intensity of ultra violet illumination, considered lines as low as 313 $\mu\mu$ to be visible. Soret [345] by photographic methods found the vitreous humor of the ox in 1 cm. thicknesses to have the same absorptive power as the cornea of 294.8 $\mu\mu$. He found that the lens of the ox absorbs rays shorter than 383 $\mu\mu$, and the entire eye has the same limit. Nevertheless he maintained that the human eye could see rays as short as 294.8 $\mu\mu$.

Eisenlohr [106] threw doubt on these results when he pointed out that fluorescence alters the ultra violet rays so that the observation by means of or in the presence of fluorescent light, is not accurate. He found even on white paper screens that fluorescence rendered rays

visible as low as 354 $\mu\mu$ in length while observation of the same light through the spectroscope showed no rays visible less than 395.6 $\mu\mu$ in length. Later Sauer [305] using metal electrodes came to the same conclusion.

With the use of the arc light de Chardonnet [67] photographed the rays able to pass through the human lens and found absorption began at the "H" line (397 $\mu\mu$) increased to the "L" line (381 $\mu\mu$) and became total at the "M" line (372 $\mu\mu$). The absorptive power of the cornea lay between the 304 $\mu\mu$ and 299 $\mu\mu$ lines and for the vitreous humor between 304 $\mu\mu$ and 310 $\mu\mu$. To de Chardonnet belongs the credit of properly emphasizing the significance of the higher absorption of the lens in determining the lower limit of visibility of the spectrum. He concluded that patients having clear media after cataract operations could see more of the spectrum than normal eyes. By thinly silvering a quartz glass plate he was able to prevent all except rays below the 343 $\mu\mu$ line from passing through so that when normal eyes attempted to observe an arc light through the plate it was entirely invisible. He found two aphakic patients however who could tell when the arc light was turned on and off or when it was moved while lighted.

Widmark [416] next took up this question in a very thorough manner. He used Hasselberg's modification of Rowland's spectroscope using a grating with a radius of 1.6 meters. The light source was an arc light with iron poles. The discontinuous spectrum obtained in this way gives sharp well known lines to examine and is free from aberrant light. Widmark examined eight aphakic patients ranging from 59 to 68 years of age. Seven could see lower in the scale than he, himself, could. Four of these were examined roughly by observing the spectrum thrown on a screen. One of these could see no better than himself so that the above more accurate method was used on the second four giving the following results for the ultra violet limit,— 313 $\mu\mu$, 313 $\mu\mu$, 342 $\mu\mu$ and 344.5 $\mu\mu$.

In order to test the normal range at various ages for comparison he examined 59 individuals ranging from 11 yrs. to 74 yrs. of age.

The results are here tabulated.

No. of Individuals	Age in years	Lower limit of vision in $\mu\mu$
10	11–20	378–395 Average = 386
14	20–30	371–395 " = 382.5
6	30–40	372–393 " = 388.9
13	40–50	380–394.5 " = 388.7
3	50–60	378–402 " = 391.7
10	62–74	379–410.8 " = 401.8

After 55 years of age, only one out of 12 individuals could see rays below 395 $\mu\mu$, that is, in the ultra violet region. The only medium which changes at that age is the lens, so that it is still more definitely proven that the lens establishes the lower limit of spectral vision in the human eye.

Birch-Hirschfeld [35] compared the visual threshold or distinguishing power, for various intensities of the same wave length, in the ultra violet region, of the aphakic eye and the eyes of individuals ranging from 14 to 70 years of age. He was thus determining not the ultra violet limit of vision, but the intensity at which a definite wave length which both groups of eyes could see, would become visible. He found that the threshold of the lensless eye exceeded that of the normal eye not inconsiderably, except in the case of a red blind physician who had developed a power of distinguishing small intensity changes that almost equalled that for the lensless eye. As the wave lengths of the light used were diminished, he found that the lensless eye gradually gained more advantage until near 381 $\mu\mu$ and below it showed far greater sensitiveness than the normal eye. His results thus agreed with those of Widmark, though the same accuracy was not attempted, — (the screen method was used) since his point was only to show the relatively greater sensitiveness of the lensless eye, in the short wave than in the long wave length ultra violet regions of the spectrum.

The appearance of the ultra violet spectrum has been variously described. Helmholtz characterized it as deep indigo blue under weak illumination to silvery blue under stronger illumination. Sekulic [334] and Sauer [305] called it silver gray in color. Widmark's aphakic patients described the first part, 340 $\mu\mu$ to 370 $\mu\mu$, as blue or violet and below that all described it as a weak light gray. More recently Schanz and Stockhausen [318], Birch-Hirschfeld [31], and others agree on lavender-gray as the best descriptive term.

The question as to how this sensation is produced, whether by direct stimulation of the retina or by the intermediation of the phenomenon of fluorescence of the lens or retina, has been variously answered. Soret [343] favored the latter view, but the work of Widmark [416] and others shows that when the lens, which fluoresces more than any other part of the eye, is removed, still greater range of vision in the ultra violet region is obtained. Further as pointed out by Widmark [416] and Mascart [240] the ultra violet region appears in sharp lines and bundles, not as a blur of light impossible to focus such as would come from the fluorescing lens. Nor could the fluorescence of the retina make these rays visible and still give a sharp image. Thus according

to Tigerstedt[366] it is generally accepted that the retina is sensitive to such ultra violet rays, as are able to penetrate the eye media and be focussed upon it.

The same does not hold for ultra red rays although Helmholtz thought it did from the work of Brüche and Knoblauch[59] in 1846 who found that heat from the Argand burner did not penetrate the eye appreciably. Cima[73], 1852, also Janssen[198], and Franz[121] in 1862, found only about 9% of the heat from a Locatelli lamp was transmitted. This was confirmed by Klug[204] in 1878 with gas and sunlight. Tyndall[371], 1865, with a 650 c. p. arc lamp found that about $\frac{1}{5}$ of the dark heat rays were transmitted through the vitreous of the ox. Engelman[110] in 1882 using the bacterium photometricum,— which always migrates to the infra red region when exposed to the spectrum, as an indicator, found the same phenomena when water glass vitreous lens or cornea was interposed. Hertel[173] in 1911 showed the lower limit of subjective and of objective stimulation of the retina were about the same, lying between 820 $\mu\mu$ and 840 $\mu\mu$. Vogt[401] however, in 1912 showed conclusively that a great amount of the ultra red light reaching the retina is not visible, in fact as much as 80% or more. Further on normal human eyes he found that 3% of the heat reached the retina and less than 1% passed on into the orbit. 20% to 25% passed through cornea or sclerotic. The aqueous absorbed 20–30 % of the heat transmitted by the cornea. The cornea iris and lens together transmit 6% of the heat falling on the cornea. The lens absorbs 30% of the heat transmitted by the cornea and iris. Vitreous absorbs nearly 60% of the heat falling on its anterior surface. The upper lid transmits 6%.

Fluorescence has long been a subject of much interest and study. A. von Graefe knew that fluorescence of the lens was due to ultra violet light and Helmholtz[160] after an extended study of the fluorescence of the lens, quinine sulphate solutions and other fluorescing bodies, concluded that fluorescence in general is due to the appearance of rays of various length, and is therefore really mixed or white light. Fluorescence was then the result of a transformation of ultra violet rays to rays of greater wave length. He considered the rays between 400 $\mu\mu$ and 300 $\mu\mu$, to be chiefly the ones transformed. Widmark noted an apparent decrease of fluorescing power of the lens as the age of the individual increased and the absorptive power increased.

Schanz and Stockhausen[315] in 1909 took up the question of fluorescence in connection with their study of the properties of "Euphosglas" which they found in certain grades to absorb rays below 400 $\mu\mu$

and still cut down the visible spectrum but little — 5% for a thickness of 10 mm. They found that fluorescence of the rabbit's lens was not diminished by 18 mm. of plate glass, therefore rays less than 300 $\mu\mu$ could not be held responsible for the fluorescence. Nor did flint glass absorbing to 350 $\mu\mu$ decrease the phenomenon but when Euphos glas was interposed the fluorescence was stopped. Therefore they limited the range of fluorescing rays to 350 $\mu\mu$ to 400 $\mu\mu$. If they allowed the light to traverse both a blue uviol glass and a Euphos glas before striking the rabbit's eye there was no fluorescence of the lens. Now when the Euphos glas only was removed after adaptation had taken place, much lid-spasm and blinking of the rabbit's eyes took place as the fluorescence began. They laid great stress on this occurrence as an indication of the painful and injurious effect of this group of rays, 350 $\mu\mu$ to 400 $\mu\mu$, on the retina. From a study of the spectrum in this region photographed through the whole eye media and from the appearance of the fluorescence itself, they came at this time to the conclusion that fluorescence was due not as Helmholtz said to transformation of short wave ultra violet light to longer waves of different length in the visible field, but due to the appearance of a new spectral color lavender-gray of definite wave length. Not only did they consider fluorescence, as did Widmark, to decrease with age but also with length of time of exposure, since the fresh lens from a gliomic eye of a child diminished notably in fluorescing power after exposure of a few hours. This decrease of fluorescing power they attributed to some breaking down or change in chemical composition which they supposed, without analytical proof, to be involved in the production of fluorescence.

Birch-Hirschfeld[38] at once objected to these conclusions, considering that nothing had been offered as real proof against the Helmholtz theory of fluorescence. He maintained that nothing had been shown as to the nature of fluorescence and even questioned whether the range of rays responsible could be established in the manner described. The fact that lid-spasm was elicited as described he could not consider as evidence of a retinal injury, since it is well known that such reflexes are easily produced by harmless light on the dark adapted eye. Aside, then, from injury to the retina by the range of rays mentioned, a mere change of intensity would account for the lid-spasm since Helmholtz has shown that fluorescent light is many times as intense, physiologically, as the light producing it. As to the diminution of fluorescence with age Birch-Hirschfeld considered the question still open, since he found the fluorescing power of the lens of an individual 70 years old

undiminished. Further he regarded fluorescence of the nature of a catalytic chemical reaction if it was to be considered chemical at all, emphasizing the fact that no chemical basis for this phenomenon had ever been established. Against the proposition that fluorescence decreased with the length of time of exposure, he cited an experiment in which he exposed and fluoresced the rabbit's lens continuously for five or six hours with no noticeable change in intensity of fluorescence whatever. He objected to drawing any conclusions from the lens of a gliomic eye since the presence of a pathological condition might readily alter the properties of the lens although it was apparently normal.

Later in 1909 Schanz and Stockhausen[316] by means of Wood's light filter which absorbs the rays below 375 $\mu\mu$ were able to make further investigations on the range of fluorescing rays in the same manner previously employed. This filter inhibited the fluorescence very little so that the range most effective in producing fluorescence was placed at 375 $\mu\mu$ to 400 $\mu\mu$. The fresh clear lenses from eyes removed by tumor or absolute glaucoma were used. The absorption power of the cornea and lens in these cases were studied by photospectrographic methods with a quartz glass optical system in the usual manner. From a study of these photographs they agreed with Birch-Hirschfeld that the cornea had a greater absorption power than glass and considered that the effectual absorption amounted to 320 $\mu\mu$ as mentioned under cornea. That absorption in the lens was increased by age was further confirmed.

Schanz and Stockhausen[318] further investigated the phenomena of fluorescence with the result that they retracted their previous idea that fluorescence represents a separate spectral color lavender-gray, and returned to the theory of Helmholtz that it is made up of rays of various length as is ordinary mixed or white light. They used the crossed prism or crossed spectrum method of Newton to analyze the fluorescent light in the following manner. With a quartz glass optical system, the light from an arc light was concentrated on a prism, the resulting spectrum was rendered linear by focussing it on a screen with a cylindrical lens, axis parallel to the length of the spectrum. Now when thin layers of fresh human lens were laid on the screen in various regions of the spectrum fluorescence was noted to begin in the blue region, become more intense in the violet region, and was strongest of all in the ultra violet region between 370 $\mu\mu$ and 400 $\mu\mu$. Below 370 $\mu\mu$ fluorescence diminished slowly. The maximum point of fluorescence was at 385 $\mu\mu$. This fluorescent light was further analyzed

by a second prism placed at right angles to the first prism and parallel with the first spectrum. The second spectrum seen through the second prism showed at once that the fluorescent light was made up of rays of different length. Of these the greater portion were green waves, a less portion of blue waves, while a considerable amount of red was also present. In the light of these findings and after a further study a few months later the following tabulation of the effect of light waves on the eye was prepared:

Visible light red to green		Visible light blue to violet	Invisible ultra violet light		
I	II	III	IV	V	
760$\mu\mu$–490$\mu\mu$	490$\mu\mu$–400$\mu\mu$	400$\mu\mu$–375$\mu\mu$	375$\mu\mu$–320$\mu\mu$	320$\mu\mu$–0$\mu\mu$	
These rays proceed unchanged to the retina and are visible.	A small part increasing with age is by the lens absorbed, and is concerned in its fluorescence. Another part fluoresces the retina and the rest is seen by the retina as blue and violet.	A part fluoresces the lens. A part fluoresces the retina. A part proceeds unchanged to the light sensitive retina. Whether the appearance of the lavender-gray is due to a direct stimulation or by intermediation of fluorescence is unknown.	Take little part in fluorescing the lens. Are intensely absorbed by the lens, reaching the retina only in young eyes much weakened.	Do not penetrate through the cornea but produce outer eye trouble.	

Hallauer[152] in 1909 spectrophotographically measured the absorptive power of over 100 fresh human lenses and found it to depend mostly on individual differences of thickness, color and consistency. For young lenses, while most of the rays were absorbed at about 400 $\mu\mu$, a certain number of more or less weakened rays between 330 $\mu\mu$ and 315 $\mu\mu$ were able to pass through. The effect of severe or chronic illness in these cases was to increase the amount of all to pass through in the latter region. Also in advanced age, where the absorption lay usually between 400 $\mu\mu$ and 420 $\mu\mu$, the effect of severe reducing diseases was to reduce the absorption power to about 375 $\mu\mu$.

Martin[238] in 1912 found the absorption power of lens suspended in normal salt solution to begin at 400 $\mu\mu$ and become complete at 350 $\mu\mu$.

Injuries to the Lens.

Czerny [85] in 1867 in his blinding experiments with sun's rays noted turbidity in the lens cortex but no change in the lens capsule. Deutschman [89] repeating these experiments in 1882 got the same results. Herzog [176] in 1903 obtained similar results with the carbon arc, glass lenses and heat filters. These lens changes were undoubtedly due to the thermic action of visible rays.

Widmark [417] in his experiments on the outer eye, 1889–1892, with the 1200 c. p. arc light noted lens changes microscopically. These he did not find when ultra violet rays were screened out by a quinine sulphate solution therefore he concluded they were the etiological factor. Ogneff [259] in 1896 repeated this experiment with a 6000 to 8000 c. p. arc lamp at a distance of 50 cm. to 1 meter for 15 to 20 min. but found no lens trouble though all the conjunctival corneal and iris troubles were present.

Widmark [418] repeated his work again in 1901 using a 4000 c. p. zinc arc in much the same way with the same results. Two rabbits A & B were exposed to the same arc light at the same time. In the case A the light traversed two glass lenses separated by 5 to 6 cm. of a 10% quinine sulphate solution. The distance from arc to lenses was 13.6 cm. and light was concentrated on the dilated rabbit's eye 6 cm. beyond the lenses. Ultra violet rays and heat rays were cut out by this method and no lens changes were found. In case B the conditions were the same except that the lenses were of quartz, separated by water. In this case in addition to the usual disturbance in the outer eye and iris, the lens capsule in the pupillary area showed at first intense staining of the nuclei, mitosis, cell proliferation and destruction. There was swelling of lens fibre bundles with partial destruction. Also transudate between the cortex and capsule.

In 1904 with a $3\frac{1}{2}$ to $4\frac{1}{2}$ amp. Finsen light, Birch-Hirschfeld [38] could not get lens changes after 5 to 10 min. exposures. Hertel [168] in 26 rabbits used in the previously mentioned experiment, and also Strebel [359] failed to get lens changes, though the usual outer eye and iris changes were produced as previously observed.

Hess [174] in 1907 used a $3\frac{1}{2}$ amp. uviol mercury vapor lamp with a 65 cm. tube. He exposed 1 to 16 hours at a distance of 10 to 30 cm. The animals used were rabbits, guinea-pigs and frogs. He found outspoken lens changes as described by Widmark. These lens changes appeared about 48 hours after a 6 to 12 hour exposure. Surrounding

the central damaged area but under cover of the iris was a ring or "wall" of deeply stained cells crowded together perhaps by the swollen central cells, first damaged. These changes appeared in the pupillary region and would show regeneration as indicated by numerous mitotic figures in the course of 2 to 4 days, if no further or stronger exposure was made. He found that interposition of glass plates cutting out rays below 313 $\mu\mu$ or even 280 $\mu\mu$ prevented lens trouble. He agreed with Widmark in thinking the glass blower's cataract due to ultra violet light.

Birch-Hirschfeld [78], however, took up this point in 1909. He exposed a rabbit's eye for 5 minutes at a time on three successive days to the light from a 5 amp. arc light which traversed first a "Euphosglas" and was then concentrated with a 20 diopter common glass lens. No heat filter was used. On the 4th day he found on microscopical examination the same lens changes recorded by Widmark and Hess. Therefore he concluded that rays in the neighborhood of 400 $\mu\mu$ must be responsible, and that probably some of the shorter blue and violet rays were effective as well as the longer ultra violet rays. Further he argued that this same group of rays was in all probability responsible for the production of the glass blowers cataract and possibly also for the production of the senile cataract, though this latter he regarded as far from proven (cf. page 677).

Without experimental evidence Wenzel [418] in 1806, von Beer in 1817 and Plenk in 1877 [84] pointed out the disposition to cataract among glass blowers, and Meyhofer [245] in 1886, found the percentage to be 11.6 in glass blowers under 40 years old. These cataracts commonly began on the left side which was most severely exposed to the heat. Robinson [298] and Stein [349] later confirmed these findings.

Schanz and Stockhausen [320] in 1910, measured the quantity and quality of the radiations from the glass blowers furnace, and the temperatures to which his head was subjected at different stages of his work. By accurate spectro-photographic measurements of the light at the distance at which the glass blower worked, they found the spectrum to be especially strong in the region from 400 $\mu\mu$ to 350 $\mu\mu$, shading down to 320 $\mu\mu$ below which there were no rays. At once they considered they had the explanation why these people had lens trouble without anterior eye trouble. The worker's head was exposed to a temperature of 110 degrees C. in taking the glass from the oven and to 45 degrees C. during the process of blowing. This temperature, while it might be a factor, is not so great as that to which many iron and blast furnace workers are exposed without receiving any eye

injuries. Temperature, therefore, they thought not nearly so much to blame as the ultra violet rays from 400 $\mu\mu$ to 350 $\mu\mu$ which fluoresce the lens most strongly. The absence of rays below 320 $\mu\mu$ accounted for the absence of outer eye trouble, in answer to the question raised by Birch-Hirschfeld during the previous year. In comparing the cataracts of glass blowers and those produced artificially by the arc light, they noted as has Widmark [418], Hess [178], Cramer [81] and others, as well as Stein [349] later, that they both begin in the pupillary region, but that while the artificially produced cataracts begin usually on the anterior pole, the glass maker's cataract starts usually on the posterior pole. For the posterior polar variety no better explanation could be offered at that time than that of Cramer [81], who believed them to be the result of the greater concentration of chemical rays at that point due to the refractive power of the eye media anterior to that point. Other theories were concentration of the chamber fluids (Leber [221]) and increased venous stasis (Peters [272]). However Snell [343] in England found cataract no more common among glass blowers than among other laborers, and Robinson [298] found the percentage increasing above normal only among the finishers working with very heavy metal glasses, after long service.

In 1909, Handmann [157] submitted an extensive statistical study of senile cataracts. Hirschberg confirmed by Schulek [330] had first suggested the intense sunlight as a factor, in the country and India, and had noted the early appearance in the lower quadrant. Handman was able to prove that the senile cataract particularly of India for the most part 81% (previously given by Greene [146] as 95%), begins in the lower quadrant of the lens. This region of the lens was found to be more deeply yellow colored, and therefore had a higher absorptive power for ultra violet. The ultra violet content of light coming from the sky at such an angle as to strike this quadrant is far greater than that reflected into the eye from the broken surfaces below, thus supporting the original idea of Schwitzer [332] that abiotic rays were an etiological factor. Presumptive evidence at least was furnished to explain the senile cataract. Schanz and Stockhausen [322] at once accepted these findings as giving the key to the etiology of a large group of senile cataracts. But Birch-Hirschfeld [32] pointed out that dwellers in mountainous and snowy regions of high ultra violet content were not prone to cataract and considered that the shorter visible rays could not be excluded here as in other cases, and Hess [184] from a mathematical standpoint considered that rays not obstructed by the lids and eyelashes could not reach the lower quadrant of the lens in sufficient quantity to account for the condition.

Martin [238] in 1912, with a single high intensity exposure,— one half to two hours at a distance of one inch from a Kromayer water-cooled mercury vapor lamp, found the lens capsule changes as described by Hess. Because the interposition of a benzol cell prevented these changes they were ascribed to ultra violet light. With repeated exposures of moderate intensity without lid retractors — at a distance of 4 inches to 3 ft. at intervals of 1 to 2 weeks over a period of $2\frac{1}{2}$ to 12 months exposure times varying from 1 to 3 hours, the following changes were noted. In one rabbit of this latter series exposed every 10 days at 4 in. distance for 1 hour over a period of 3 to 12 months lens changes somewhat as described by Hess were found, but differed in that the "wall" was wider, the central cells were uninjured, and proliferation was 2 or 3 cells thick. There was present in all of this series slight corneal opacities. Others of the series, less severely exposed, had no corneal opacities or lens trouble, while those more severely exposed,— 3 hours every 2 weeks at 4 inches for 3 to 11 months,— showed dense corneal opacities which had undergone vascularization but the lenses were clear and capsule normal, supposedly protected by the dense corneal opacities.

Sharply to be distinguished from these are cataracts experimentally produced by exposure to light, are those resulting from the actual transit of the electric current through or near the eye. Leber [221] in 1882 first explained, the long noted tendency to cataract in people struck by lightning, as due to the electro-chemical reaction. This was experimentally demonstrated by Hess [117] in 1888 who made an electric spark to impinge in the supraorbital region of a rabbit. He noted a central destruction of lens epithelium and vacuolization of lens fibres with a marked secondary peripheral mitosis and proliferation of the lens epithelium resulting in the formation of equatorial cataracts. Likewise Kiribuchi [203] in 1900 using the Leyden condenser spark produced the same results. Clinically many cases of cataract after lightning stroke or short circuit have been reported as due to ultra violet rays but really must be included in this last group. Birch-Hirschfeld [38] in 1909 found, in a study of all such cases reported up to that time, injuries such as burns and cicatricial formations or impairment of nutrition or nerve supply, which would readily account for cataract formation. These cataracts are further distinguished by the fact that clinically and experimentally they do not appear centrally as do those in purely blinding experiments but peripherally. Again on an experimental basis the length of exposure time is seldom if ever long enough. No cases of lens trouble from lightning without bodily

injury have been reported. Essentially the same is true in blinding due to short-circuit arcs, though sometimes it is difficult to distinguish mechanical from light effects, as in cases reported by Grimsdale and James [198] and by Posey [282] in 1911.

The Retina: Injuries, Scotomata, Erythropsia.

That blindness may result from direct observation of the sun of eclipses, has been known without doubt for ages. Indeed Galileo [31] is known to have injured his eyes by observation of the sun with his telescope. Galen [129] cites cases of blinding, with more or less subsequent return of vision, in observers of eclipses of the sun. He also noted that central scotomata or blind spots often resulted in the same way. Reid [294] in 1761 and Soemmering [342] in 1791 according to Hess [184] probably gave the first accurate description of the subjective phenomena of sun blinding.

Less frequently the same ocular disturbances have also long been noted in seamen long exposed to strong reflection of the sun's rays from water surfaces. Likewise travelers over desert or glary plains are not uncommonly afflicted with these visual disturbances.

Czerny [85] as early as 1867 showed that a lesion of the retina of the rabbit visible with the ophthalmoscope could be produced by the sun's rays. Coccius, Ruete, in 1853, and Jaeger in 1854, had already described the ophthalmoscopic changes in the human eye. Czerny threw, by use of a concave mirror, and glass lens system concentrated sun's rays which had traveled a 20 cm. water heat filtering tube, into the eye of the rabbit for 10 to 15 sec. The region of the retinal image on exposure was found to be whitened and seared. A section under the microscope showed what he described as a coagulation of the albuminous substances of the retinal elements.

On the 17th of May, 1882, there was an eclipse of the sun which in a few days brought four cases of sun blinding into Leber's clinic in Göttingen. Deutschmann [89] at once reported them with a repetition of Czerny's experiment in which he fully confirmed Czerny's findings. One of the four cases used a smoke glass to observe through and another a blue glass, but each received severe injury nevertheless. On ophthalmoscopic examination all four cases showed a characteristic appearance of the macular region varying somewhat in degree corresponding with the degree of injury. In his experiments Deutschmann arranged a convex lens to transmit the sun's rays reflected from

a concave mirror. The distance separating the two was equal to the sum of their focal lengths so that parallel light was thrown into the atropinized eye of the rabbit. Even after a second's exposure a silvery white spot round to oval in shape, covered the retinal image region. It was surrounded by a brownish ring. Longer exposure enlarged the silvery central spot and the surrounding rings became paler and took on a silvery sheen. Microscopic section showed droplets and clumping of the coagulated albumins of the retinal cells. Surrounding and below these areas were exudative and then hyperemic areas. The choroidal pigment was increased and showed a tendency to wander. Indeed there was so much similarity to early stages of choroiditis disseminata that he was the first to consider the possibility that heat and light rays may be an etiological factor in the latter disease. However Aubaret [1900], Hess [1902], and Garten [1908] have since attempted, without success, to prove this proposition. To determine the influence of heat Deutschmann passed the rays through a tube of clear running water 20 cm. long. The changes could be produced but it always took a few minutes longer. Therefore he concluded that both heat and light are active as etiological factors. In none of these cases with short exposure times were outer eye troubles noted.

In 1892 Widmark [413] repeated Czerny's and Deutschmann's work with a 1200 c. p. arc light. The exposure time was 2–12 hours, usually 4 hours. With the 10% quinine sulphate filter to remove ultra violet light he found much less retinal trouble than with the quartz glass system. Also less effect through yellow bichromate filters than through blue, so that he concluded that blue violet and ultra violet rays were most effective.

Herzog [176] in 1903 reported that he had repeated the work of Czerny and Deutschmann in 1898 and found that the circumscribed retinal lesions could be effected in $\frac{3}{5}$ second. A similar but more diffuse retinal change could be produced in $\frac{1}{2}$ to 2 hours with a 15 amp. arc light whose rays were concentrated on the rabbit's eye after traveling a 28 cm. tube of alum water. Further similarity in action was noted in the production in two or three old rabbits of an opaque cataractous condition of the lens visible to the eye as Czerny and Deutschmann had noted. When the cornea was continuously irrigated with normal salt solution to avoid overheating, the result was a cloudy swelling of the epithelium which was less transparent than the simple desquamation that resulted without the excess of moisture. All these changes, including the cataract formation already described he ascribed to light transformation to heat, and not to ultra violet light.

However, Aubaret [6] in 1900 was inclined to disregard heating effects in sunblinding since he found a thermometer held in sunlight concentrated by 40 D diaphragmed lens, only registered 1° to 2° increase in temperature. But Birch-Hirschfeld [38] showed that 50° paraffin in thin layers on black paper was melted in a few seconds when exposed to sunlight as the retina would be in sunblinding but when white paper was used under the same conditions several minutes were required to melt the paraffin, thus demonstrating the effect of black retinal pigment in absorbing heat.

The phenomenon of light adaptation and the effect of various wave lengths of light on the retina has been studied by a number of observers, Mann [235], Kuhne [276], Pergens [269], Nagel [257], van Genderen-Stort [137], and more recently by Hess [182], Birch-Hirschfeld [39] and others. Van Genderen-Stort [137] in 1887 showed that pigment wandering in the choroid and retina was least active in yellow light and this finding called attention to the value of yellow glasses as protection for the eyes. He further amplified the knowledge of the effect of light on the finer structures of the retina of the dark adapted eye. When such an eye is exposed to light he showed that the pigment cells just outside the rods and cones send down processes, apparently between the rods, which carry with them much pigment and thus tend to isolate optically the rods from each other. At the same time the cones tend to escape this isolation somewhat by moving in the same direction towards the light. Another change found to take place was the bleaching of the visual purple found in the outer part of the rods in the dark adapted eye.

The effect of ultra violet ray and light fatigue on the finer structures of the retina has been studied by Widmark [412], Czerny [85], Deutschmann [89], Ogneff [259], Bach [14], Stebel [359], Kiribuchi [203], and Terrien [364]. Except for the last three, who found a very slight ganglion cell chromatolysis, their results were negative, which Birch-Hirschfeld [37] later thought was due to insufficient light intensities having been used. In snow blinding nothing more than hyperemia of the retina and optic disc have been reported (Reich [92], 1880).

Birch-Hirschfeld [37] in 1904 made some experiments on rabbits in which he claimed to have produced definite pathological changes by exposing the eyes to ultra violet light. In his first series of experiments he separated out the ultra violet light from a 15 ampere carbon arc light by means of a quartz prism, and obtained retinal changes only in eyes from which he had removed the lenses. In his second series he used the direct light from a 3 to 4.5 ampere water cooled Finsen

iron arc and obtained well marked changes, notably chromatolysis and vacuolization of the ganglion cells, in lens containing eyes as well as in two aphakic eyes. These experiments are described and discussed in detail on page 687.

From the foregoing experiments Birch-Hirschfeld concluded that the lens did not afford complete protection to the retina from the specific action of ultra violet light. Best [27], however, took the view that no danger to the retina was to be feared from ultra violet light since he found he was able to look at the sun directly without deleterious effects for ten seconds through a blue uviol glass which transmits freely the ultra violet waves from the sun, but absorbs all visible waves longer than about 470 $\mu\mu$ in length.

Birch-Hirschfeld [35] in 1908 reported five cases of visual disturbance among workers in mercury vapor illumination. He concluded that after long occupation with unprotected eyes in mercury vapor arc-light a disturbance of retinal function may result with or without an electric ophthalmia or a similar conjunctival reaction. These injuries took the form of pericentral scotomata for red and green. By the aid of a Priestly Smith scotometer these scotomata were mapped out. The affected region had a sector or ring-formed shape at a distance of 15 degrees to 20 degrees from the fixation point. Red usually appeared yellowish and green as a gray or even white. The central color vision was only in two cases injured in the sense of red-green blindness, though floating spots often temporarily obscured the fixation point. The color scotomata disappeared in the course of a few weeks if protecting glasses were worn or if work in ultra violet light was abandoned.

The solar eclipse seen in Europe on the 17th of April, 1912, afforded excellent opportunities for observation of ophthalmoscopic and functional retinal changes due to sun blinding. In the series of 50 reported by Birch-Hirschfeld [41], four cases showed normal eye grounds, 19 others had increased foveal reflex, with frequently a concentric irregular reddish-brown area, which in eleven cases cleared up at the end of a week. In 16 other cases there was noted irregular pigmentation of the macula and small gray puntiform spots or globules which remained unaltered for months. In 31 cases an absolute central and in 19 cases an absolute paracentral scotoma was found which afterwards became relative scotomas. The rest had relative central scotomas to begin with. These were mostly eccentric downward extending 1° to 10° The majority of cases with the milder injuries regained almost normal visual acuity in the course of a few weeks. In small numbers of single

cases numerous previous observers [184], Vinsonneau, Stocke, Pergens v. Pflugk, Arlt, Lescarret, Menacho, Villard, found similar changes. Lamhofer in 1912 found a chorioretinal exudate with pigmentation and at the same time Best reported a case with decreased peripheral field and adaptation. Erythropsia has also been noted by Birch-Hirschfeld and by Braunschweig.

In 26 out of 36 cases of sun blinding in the eclipse of 1912, Jess [200] working in Hess's clinic found a ring-scotoma 20° to 40° from the fixation point. In this ring formed area, white appeared gray, and colors were not seen in a few cases, in others red was called yellowish, green was called gray, and blue was called yellow. In the course of a week the damage gradually decreased until only a small semicircular area of scotoma was left below. Hess [184] states that this finding has been verified by Peppmueller, Pergens, and Hoppe. Speleer found enlargement of the blind-spot ring-scotoma and concentric contraction of the field after sun blinding. Birsch-Hirschfeld [43], however, has taken exception to the ring-scotoma of Jess, finding that it was a normal phenomenon and not specially related to eclipse-blindness.

The large number of functional impairments due to lightning and short-circuit flashes cannot be referred definitely to any given range of wave lengths. These derangements have been reported in great numbers and variations. There may be permanent blindness or temporary blindness in one or both eyes. Central and peripheral scotomata are more common and while usually temporary they may be permanent. Hancock [156] in 1907 reported a case of ring-scotoma. Disturbances of color vision are very common but temporary. These include erythropsia, red blindness, red green and blue green blindness and scotomata. Ophthalmoscopic examination may show nothing even in severe cases or there may be punctate spots in the macula as in sun blinding, Uhthoff [375], Haab [51], Terrien [365]. Birch-Hirschfeld [31] considers rays from 350 $\mu\mu$ to 400 $\mu\mu$ most active in producing these disturbances with probably even more assistance from blue and violet rays because of the greater intensity.

While usually there is outer eye trouble in these cases a few have reported functional disturbances without photophthalmia. Nelson Dering [184] Le Roux and Renaud [225] Maclean [234] and Purtscher [289].

In snow blinding functional disturbances have been noted to take the form of temporary amblyopia (Widmark [412] citing Enald) night-blindness (Widmark [412]) and day blindness. In 1907 Best & Haenel [29] found after snowblinding a central scotoma for red and green extending about 10° from the fixation point. This disturbance disappeared in the course of six weeks.

Very recently Behr[17] has found marked reduction in the light adaptation power in four patients complaining of visual disturbances after continued work by arc light and strong incandescent lights. They noted that after working by these lights and then moving to a darker part of the room that they could hardly see to work at all, or on coming out into sunlight everything appeared gray or dark. In twilight or after a rest in darkness the vision improved slowly only to receive another setback on further exposure to the artificial light. These patients were tested on the Piper instrument for measuring the dark adaptation power. Normally as Piper has shown there is a slow increase of sensitiveness during the first 61 to 10 min. in the dark and then a sudden marked increase in 30 to 35 min. followed by a further slow increase until after 45 min. the maximum light perception sensitiveness is reached. Behr found in none of these cases was there much increase of sensitiveness after 10 min. and at the end of 45 min. was still $\frac{1}{8}$ to $\frac{1}{7}$ of normal. These cases were advised to work less by strong artificial lights and Euphos glas was prescribed. Mild light such as oil lamplight was advised where possible. Three of the patients were presumably cured because they made no further complaint, while the fourth whose case was followed, regained in a short time a light adaptation that was better than normal.

Erythropsia or Red Vision.

Hildige[171] in 1861 noted erythropsia in snow blinding. Mayerhausen[241] and Steiner[350] in 1882 reported cases after blinding by lightning and short circuiting and by the sun's rays both directly and indirectly as from water surfaces or from snow. Cases which occurred after cataract operations were further reported by Dimmer[92] and Putscher[287] in 1883. Widmark in his correlation of electric ophthalmia and snow blinding noted that erythropsia occurred in both but did not investigate the subject further. Fuchs[126] in his monograph of 1896 emphasized the rôle of ultra violet light in all these cases especially in cases of snow blinding, aphakia, and electric ophthalmia. Using the fact that established by Kuhne[216] and Konig two years before, that the visual purple is most rapidly bleached by rays below 500 $\mu\mu$. Fuchs advanced the theory that the regenerating visual purple accounted for the red vision as an entopic phenomenon. Schulek[329] produced Erythropsia by observing spectral ultra violet

light, also Birch-Hirschfeld did the same while working with the Schott-light. However, Vogt [397] in 1908 dilated his own pupil and after producing erythropsia by observing a sunlit snow field found no decrease in the red vision when he went into a room illuminated only by a light whose red rays had all been screened out by an Erioviridin filter. Vogt [397] in 1908 and also Wydler [422] in 1912 considered erythropsia as the red phase of the after picture of the intense white surface. Best [27] in 1909 agreed with Vogt's view and considered erythropsia due to visible rays since he could produce it by looking at a snow surface through a yellow glass cutting out rays below 400 $\mu\mu$ but not with a blue uviol glass. However Birch-Hirschfeld [31] could not consider that ultra violet rays were relieved of responsibility entirely since the wide pupil alone of the aphakic eye could hardly account for erythropsia, as Vogt held, by admitting a greater quantity of light. He therefore concluded that invisible as well as visible rays were active in the etiology of erythropsia.

Rivers [296] in 1901, advanced a theory as to the red color based on an observation made by Brüche in 1851 that the eye under normal conditions is more or less completely adapted for red. Rivers attributes the color in erythropsia to the blood in the anterior retinal layers. Schoute [184] objected to this theory on the basis that Purkinje's experiment of the entopic vision of retinal vessels shows that light is absorbed by the blood and they give dark shadows.

Protective Glasses.

In 1900 Schulek [330] first studied the means of protecting the eyes against ultra violet rays and found that solutions of Triphenylmethane in xylol and Nitrobenzol in Alcohol had the highest absorptive power of the transparent media examined. These liquids absorbed practically all rays below 396 $\mu\mu$. He suggested that these solutions be enclosed in flat oval shaped glass chambers made to fit the eyes and to protect them from injuries due to ultra violet radiations.

Stearkle [348], Vogt [396] and Hallauer [153] studied the absorptive properties of blue uviol, yellowish and smoky gray glasses. The last named worker produced a glass mixture by a secret process the so called "Hallauerglas." After a similar study, Fieuzal [119] produced in like manner Fieuzelglass, which however, was not greatly used on account of its color. Also a yellow-green glass patented under the

name of Enixanthosglas was offered as well as a variety of the modifications by glass manufacturers. Here again the color was not satisfactory.

Schanz and Stockhausen[309], after finding that electric ophthalmia could be produced through 18 mm. of common glass as has been mentioned, began to study glass manufacture in the hope of producing a colorless glass of high ultra violet absorption power for general use. In 1909 they produced and patented a glass of higher absorptive power than hard flint and called it "Euphosglas." At first it was made in grades 1, 2, 3, and 4, but recently other grades have been added. It has a light yellowish green tinge and fluoresces in ultra violet light.

Birch-Hirschfeld[38] in 1909 studied photometrically the absorption power of these and other glasses with considerable accuracy. In the same year Vogt[304] compared a new and very hard flint glass produced by Schott with his absorptive solutions and was surprised to find that it had about the same efficiency, beginning to absorb at 405 $\mu\mu$ and giving practically complete absorption below 396 $\mu\mu$.

In 1909 Hallauer[155] measured photometrically the absorptive power of the various protective glasses available at that time. The thickness varied from 1 to 3 mm. and exposure time of one minute. The average results follow.

Common glass	absorbs	to	295 $\mu\mu$
Blue glass	"	"	300
Lead glass	"	"	305
Smokey gray	"	"	325
"Gonin" glass	"	"	330
Schott's heavy flint	"	"	340
Fieuzal yellow glass	"	"	375
Enixanthos glass	"	"	380
Euphos gray glass	"	"	390
Euphos green glass	"	"	390
Hallauer glass #64	"	"	420

As glasses became available to cut out various wave lengths the question arose as to what spectral range constituted the best illumination.

Voege's[388] answer to this question in 1908 raised a considerable controversy. He maintained that the light from the clouds or clear sky had been for ages the normal illumination for the eyes, but nevertheless contained a considerable amount of ultra violet light as low as

300 $\mu\mu$ in wave length. He examined the spectra of various high power arc lights with opal and milk glass shades or coverings. These spectra compared very closely with that of cloud light.

Hertel and Henker [172] in support of this view carried out a very accurate set of measurements, in the laboratory of C. Zeiss, Jena, of the cloud and skylight spectrum, of variously covered or shaded high power arc lights and of the percentage of penetrability and absorption power of various glasses for wave lengths in different parts of the spectrum. The spectrum of the clouds or clear sky was found to contain no rays below 300 $\mu\mu$ and very few below 310 $\mu\mu$.

Having what they considered an ideal light the next question was in what manner should the artificial lights be compared with it. In producing injurious effects on animals they noted that unprotected lights of very great intensity had been used under conditions never found in modern lighting systems therefore previous observers had studied entirely atypical conditions and not the conditions to which human eyes are really exposed. Widmark had used a 1200–4000 c. p. arc light without covering at a distance of 25 cm. from the animal's eye for length of time ranging from 2–4 hours and longer. Likewise Birch-Hirschfeld had used similar arc lights with and without dispersion through a prism usually at a distance of 10 cm. to 20 cm. Hess had used a Schott uviol lamp 65 cm. long of 3–3½ amp. The animals were at a distance of 10 cm. to 20 cm. and the time of exposure 1–16 hours. These data represented the average experimental conditions necessary to produce injuries but not at all the conditions to which mankind is at present exposed.

Therefore they considered a far better criterion for practical purposes would be accurately to photograph the spectrum of arc lights at the minimum distance of actual service in lighting, and to find whether or not such globes or mantles can be placed around the light source as to render the spectrum, in quantity and quality, within the range of the cloud or skylight spectrum.

Accordingly the lights were placed 50 cm. to 100 cm. distant from the spectroscope opening. The optical system of the spectroscope was made of quartz glass. The effect of indirect illumination and of half spherical or inverted bowl shaped shades for increasing horizontal illumination, was also measured by raising the light and its shade about 40 cm. above the axis of the spectroscope condensor.

In all cases under these conditions by use of the usual milk glass and opal glass shades they had no difficulty in getting a spectrum comparing in quantity and quality very closely with the spectrum of

cloud light and entirely within the spectrum obtained from ordinary Welsbach gas mantle lights with ordinary clear glass shades. They used a variety of lights including flaming arc and mercury vapor arc lights.

The absorptive power of various other glasses proposed for this purpose, by their inventors was determined by use of the most accurate available method. In the visible part of the spectrum the absorptive power was determined by use of a polarizing spectrophotometer with crossed Nicol's prisms, while in the ultra violet region the same instrument was used with optical parts of quartz glass. The results are tabulated below in terms of percentage of penetrability of various wave lengths. The absorption power is obtained by simply subtracting these results from 100. In all these cases a thickness of glass of 1 mm. was used indicated by A′, except in the case of the very dense Neutralglas which was measured in thicknesses of 0.1 mm. indicated by $A^{0.1}$.

Concerning protective glasses for the eyes, Hertel and Henker believe the same criterion should be followed, namely, that the best glass is the one which will reduce the spectrum of the particular light to which the eyes are exposed to the closest possible approximation to the spectrum of cloud and sky light. For observation of the strongest arc lights at close range, the condition under which certain workmen are placed, they consider the Neutralglas F 3815, of Schott to be the best. With this glass, in layers thinner than any other glass, one may observe directly the bare 20 amp. arc light at 50 cm. without injury since the spectrum is about the same as that of cloud light minus the ultra violet portion. The thickness of Hallauerglas No. 64 necessary to give the same results as Neutralglas of 0.8 mm., was 9.0 mm.; and of Euphosglas No. 4 was 38 mm.

Next to Neutral glas, for this purpose stood the smoky or Rauchglas No. 276 and Sonnenglas No. 66 of the Fredener glass works. After these came Hallauerglas No. 66, while Hallauerglas No. 62 and No. 64 and Euphosglas Nos. 1, 2, 3 and 4, were not strong enough in absorptive power.

Schanz and Stockhausen[321] at once criticised the above work, objecting particularly to the premises on which the decisions were based, namely that skylight or cloud light can be taken as the ideal light. Against this view was cited particularly the work of Handmann showing that a very large group of cataracts begin in the quadrant of the lens most exposed to the light of the sky during life. Since it was definitely known and admitted by all that certain injuries to

PERCENTAGE OF PENETRABILITY.

Wave length in $\mu\mu$		644	578	546	509	480	436	405	384	366	341	332	313	309	280
Euphos-Hellglas No.	1A [1]	88	93	93	90	88	79	78	52	37	39	24		8.2	.56
	2 "	86	94	93	92	82	54	27	2.3	.38	.72	1.8		6.2	
	3 "	83	89	86	80	74	42	15	8.0	.24	48	57		2.8	
	4 "	82	90	85	84	71	25	32	.49	.40	31	45		1.9	
Hallauer Glas	62 "	89	100	100	89	86	83	96	84.	81.	46	1.8	24		
" "	64 "	85	62	59	52	33	6.4	.12	1.7	6.7	16	43	62	27	6.8
" "	66 "	33	37	35	27	13	.5	34.	4.3	0.1	4.3	27	48	13	1.4
Gelbglas F	4313 "	98	97	93	83	9.4	2.1	1.2	0.1						
Neutralglas F	3815A$^{0.1}$	35	35	37	35	34	30.	15.	5.9	1.8					

the eye could be produced by ultra violet light, any light containing these rays could not be considered ideal light. The positive findings, previously mentioned of pathological changes due to ultra violet light, violet and perhaps blue rays, such as the glass blower's cataract, erythropsia, blindness permanent and temporary, and scotomata as well as other functional retinal disturbances, they held to be against the assumption that light containing considerable amounts of ultra violet, violet and blue rays, can be considered ideal and harmless. Further minor objections were made to the exposure time used in photographing the different light sources and it was pointed out that their newer glasses "Euphosglas A" and "Euphosglas B," more suitable for technical purposes, had not been examined.

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FIGURE 1. Exp. 69. Magnetite arc, double lens system, no screen, exposure 20 minutes. This exposure was about 200 times the liminal exposure for photophthalmia.

Cornea 12 days after exposure. The epithelium has reformed. The stroma is softened down to Descemet's membrane. The unevenness of the corneal surface is due to the irregular shrinkage of the semiliquefied tissue in the process of fixation. Note that the effect on the stroma is progressively greater and more widespread towards the external surface. Photo. × 12.

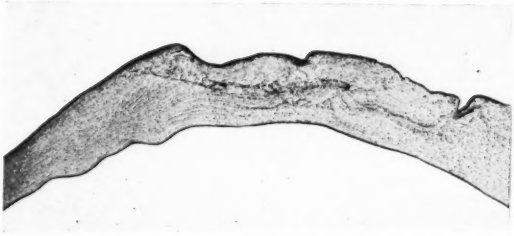


Fig. 1.

FIGURE 2. Exp. 81. Magnetite arc. Double lens system, water cell, flint glass screen (305 $\mu\mu$), exposure 1 hour.

Relatively slight heat effect on the cornea after three days. The epithelium is intact but the endothelium is destroyed. The corneal corpuscles are present and actively proliferating in the anterior layers, while many of them are destroyed in the posterior layers. Photo. × 32.

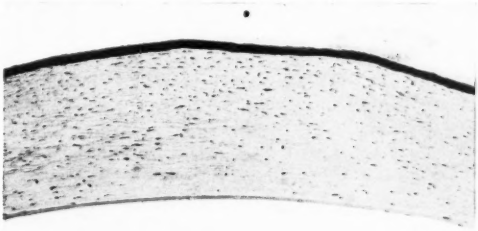


Fig. 2

FIGURE 3. Exp. 88. Magnetite arc. Double lens system, no water cell. Flint glass screen (315 $\mu\mu$), exposure 1 hour.

Marked heat effect on cornea after 48 hours. The epithelium is intact. The stroma is swollen to twice its normal thickness and the corpuscles are completely destroyed in the most exposed area. At the periphery the reappearance of the corpuscles is abrupt and they are in active proliferation. The endothelium is destroyed over a large area. Photo. × 39.

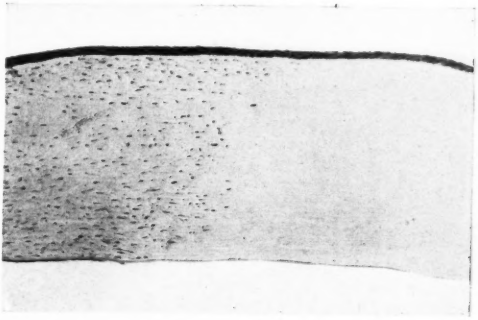


Fig. 3.

PLATE 2.

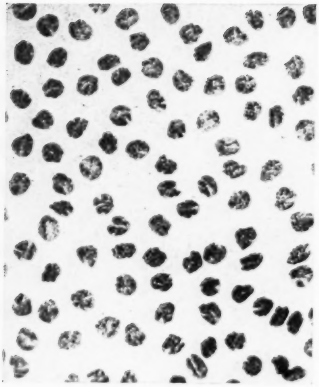


Fig. 4.

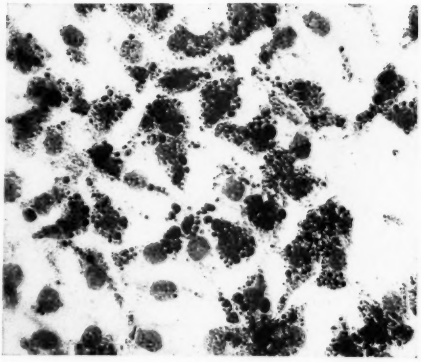


Fig. 5.

FIGURE 4. Normal lens capsular epithelium of rabbit. Flat preparation. Photo. × 264.

FIGURE 5. Exp. 55. Magnetite arc. Single lens system, crown glass screen (295 $\mu\mu$), exposure 1 hour.

Lens capsular epithelium 24 hours after exposure, showing marked abiotic effects. The cells are swollen and most of them contain granules. In the photograph the basophilic and eosinophilic granules cannot readily be distinguished from each other. Photo. × 264.

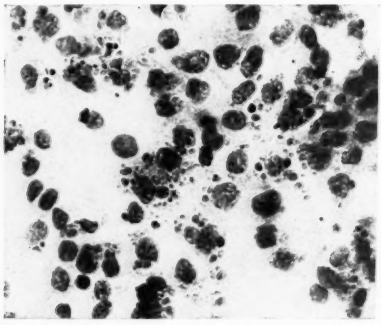


Fig. 6.

FIGURE 6. Exp. 67. Magnetite arc. Double lens system, no screen, exposure 20 minutes, about 200 times the liminal exposure for photophthalmia.

Lens capsular epithelium 48 hours after exposure, showing marked abiotic effects. The darker granules in the cells are basophilic, the lighter, eosinophilic. The clear spaces in this and the previous figure are due to some of the cells having failed to adhere to the capsule. Photo. × 264.

PLATE 3.

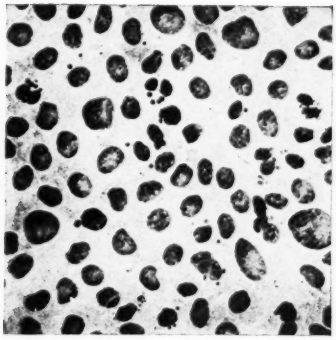


Fig. 7.

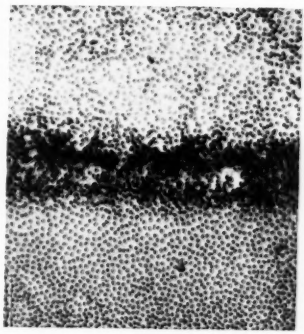


Fig. 8.

FIGURE 7. Exp. 70. Magnetite arc. Double lens system, no screen, exposure 20 minutes.

Lens capsular epithelium 2 months after exposure, showing reparative changes. The nuclei vary greatly in size and many of them are extremely large. Some of the cells contain double nuclei. The small dots in the cells are nuclear buds constricted off from the main nuclei. They are not related in any way to the granules in Figs. 3 and 4. Photo. × 264.

FIGURE 8. Magnetite arc. Double lens system. Crown glass screen (295 $\mu\mu$), exposure 20 minutes.

Lens capsular epithelium 19 hours after exposure, showing wall of deeply staining cells, corresponding in position to the pupillary margin. Photo. × 42.

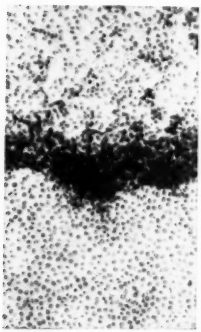


Fig. 9.

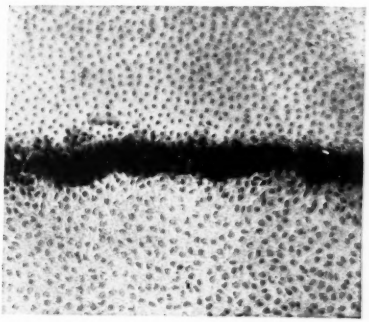


Fig. 10.

FIGURE 9. Exp. 54. Magnetite arc. Single lens system, crown glass screen (295 $\mu\mu$), exposure 20 minutes.

Lens capsular epithelium 48 hours after exposure, showing wall. Many of the cells in the unexposed zone outside the wall are in mitosis. Photo. × 42.

FIGURE 10. Lens capsular epithelium 48 hours after injection of Lugol's solution in the anterior chamber, showing wall similar to that produced by abiotic radiations. (See page 676). Photo. × 106.

PLATE 4.

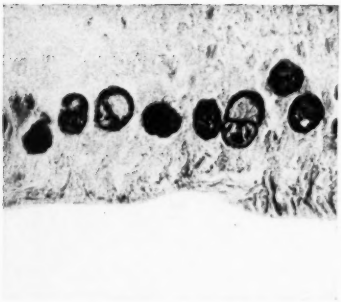


Fig. 11.

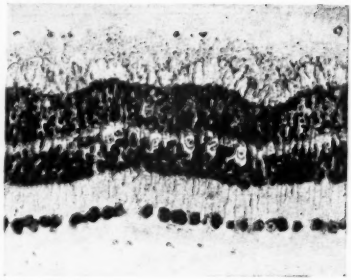


Fig. 13.

Fig. 12.

FIGURE 11. Exp. 78. Magnetite arc. Double lens system, water cell, flint glass screen (298 $\mu\mu$), exposure 1 hour.

Showing retinal ganglion cells of rabbit unaffected 48 hours after exposure. Thionin stain. Photo. × 706.

FIGURE 12. Exp. 53. Magnetite arc. Single lens system, water cell, crown glass screen (295 $\mu\mu$), exposure 12 minutes.

Heat effect on retina after 48 hours. In the affected area the rods and cones are disintegrated and the nuclei of the nuclear layer are fragmented. The inner layers of the retina are normal. Photo. × 42.

FIGURE 13. Same experiment. Affected portion of retina under higher magnification. Photo. × 190.

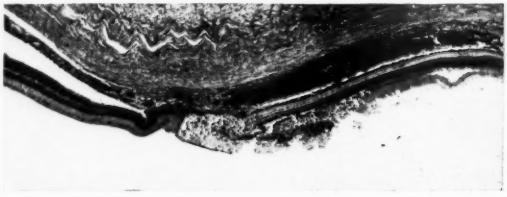
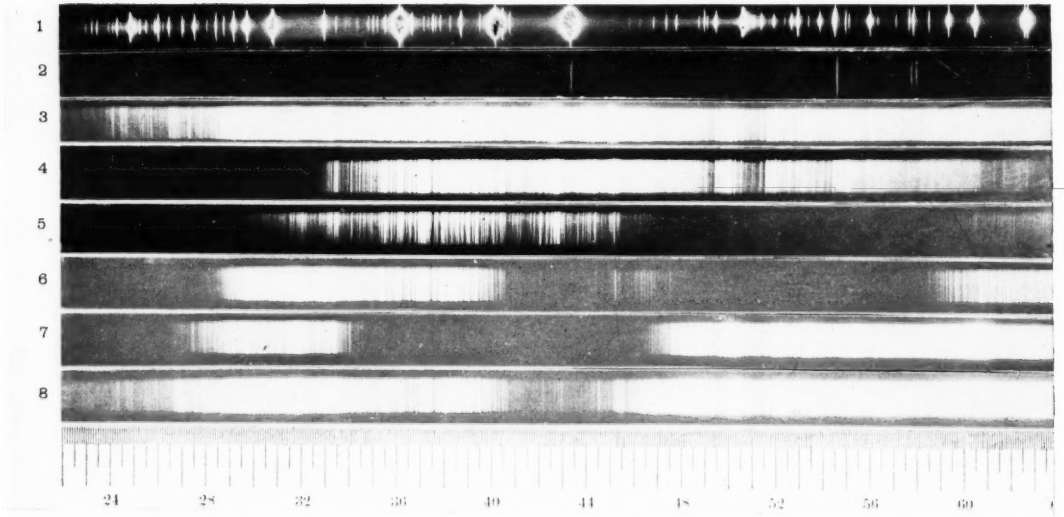


Fig. 14.

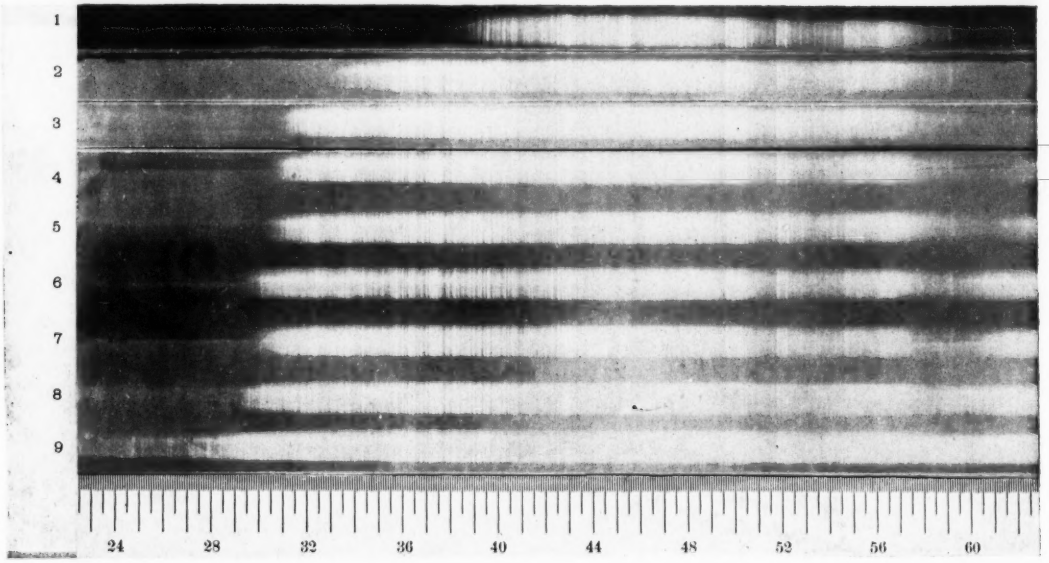
FIGURE 14. Exp. 98. Sunlight concentrated by large mirror. Water cell, no screen. Exposure 14 seconds.

Intense heat effect on retina after six days. On the left, the retina is normal. On the right, the outer layers including the rods and cones are coagulated and retain their forms, while the inner layers are disintegrated, the heat here having been insufficient to coagulate them. For the same reason the entire thickness of the retina is disintegrated at the margin of the affected area. The choroid shows a large hemorrhagic extravasation. Photo. × 28.

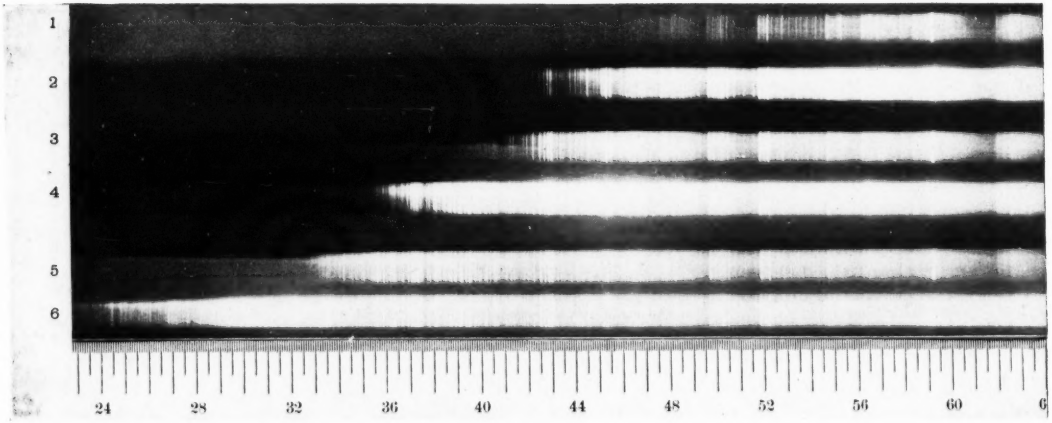
PLATE 5.



1. Hg (2nd order overlaps on right). 2. Hg (Chief visible lines). 3. Magnetite arc. 4. $CuCl_2$ 1.5%. 5. Uviol Glass (Blue, 2 mm.). 6. Blue Uviol 1 mm. + Auramin 0.0007% in H_2O. 7. Auramin 0.0015% in H_2O. 8. Auramin 0.0007% in H_2O.



1. Euphos #1; 2. Flint, $\mu_D = 1.69$; 3. Flint, $\mu_D = 1.61$, 2 mm.; 4. Do. 1 mm.; 5. Flint, $\mu_D = 1.57$; 6. Flint, $\mu_D = 1.54$; 7. Flint similar to 6; 8. Crown $\mu_D = 1.51$; 9. Magnetite arc. All discs 2 mm. thick except 4. (See page 647.)



1. Deep yellow-green; 2. Deep amber; 3. Hygat, dark; 4. Hygat, medium; 5. Hygat, light; 6. Magnetite arc. All discs 2 mm. thick. (See page 747.)

PLATE 8.

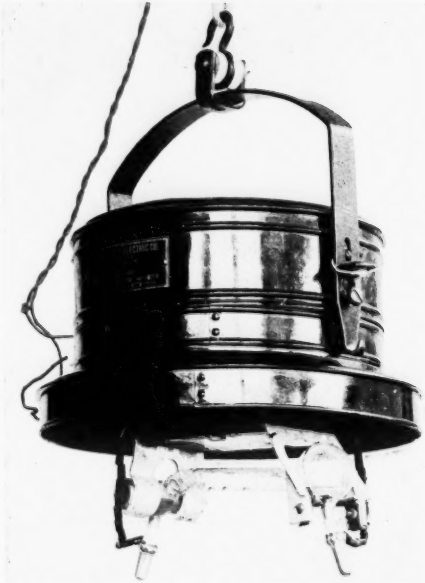


FIGURE 1. Quartz Mercury Lamp. (See page 637.)

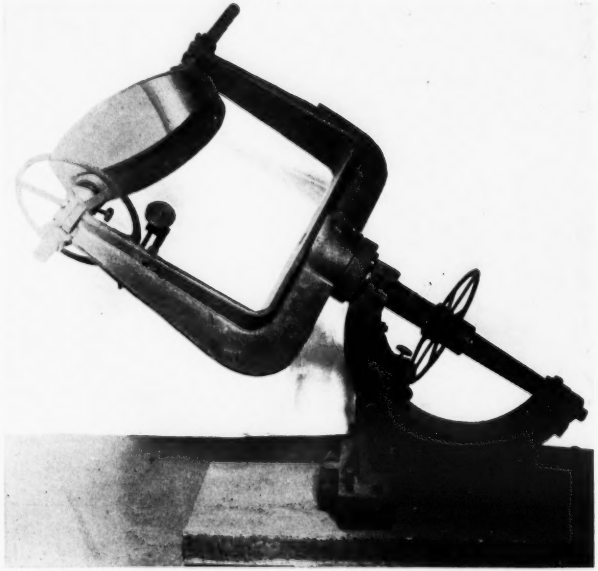


FIGURE 2. Concave Mirror and Mount. (See page 721.)